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Orthopedic Supplement
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Closed Reduction Versus Open Reduction of Fractures

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HISTORY

Judging from the history of orthopedics, man has had considerable difficulty in utilizing good judgment and adequate skill in the care of fractures. With the introduction of cannon shots at the battle of Crécy in 1346, new problems arose. The ligation of major blood vessels, introduced by Paré during the sixteenth century, helped solve some of the problems that man had created. Joseph Lister in the 1800's, using his carbolic acid spray and antiseptic technic, opened another dark cavity of orthopedic surgery, allowing light to enter. In 1852, Mathijsen, a Flemish army surgeon, introduced plaster of Paris bandages as substitutes for braces and splints in the immobilization of skeletal fractures.[1] The importance of Mathijsen's contribution cannot be exaggerated. Of recent contributions, the greatest and most outstanding by far was the introduction of sulfonamides in 1935 by Domagk.[1] The use of sulfonamides permits a wider choice of therapy for the fracture case.

Although open reduction of fractures was apparently attempted many times in the past, Lapuyade[1] and Sicre in 1775 are credited with the first written account; they used wire as a means of internal fixation.[1] Sir William Arbuthnot Lane, 1865-1943, used plates and screws quite successfully as a means of internal fixation and introduced no-touch technics. His visit to the United States in 1910 stimulated American surgeons to become interested in his methods.[1] Americans, being notorious gadgeteers and always in a hurry, seemed to take to open reduction as the proverbial duck to water. It is unfortunate that so many of us do not bother to measure the depth, the swiftness of the current, or our ability to swim before plunging into the water.

Even with gentle handling of osseous tissue, no-touch technic, 24-hour skin preparation prior to surgery, antibiotic therapy before and after surgery, and thorough debridement and cleaning of the compound fracture, infection remains the greatest complication of open reduction. My aim in this brief review is to stimulate you to an objective review of orthopedic history and to an equally objective review of the fracture technic you are employing in your own practice today.

BLOOD SUPPLY AND COMPOSITION OF BONES

Without adequate circulation, life of bone or any tissue ceases. In a long bone the epiphysis has its own set of perforating vessels. It has no communicating or anastomosing branches with the metaphysics in the juvenile and, at best, only possible anastamosis after the epiphyseal plate has been closed in the adult. The metaphysis also has its own blood supply in the form of perforating vessels and, like the epiphysis, seems to be abundantly supplied. In the diaphysis this supply is somewhat meager by comparison. The nutrient artery enters at the midshaft and then bends toward the center of ossification that first joins with the shaft. This supplies the endosteum, the medullary canal, and the inner part of the cortex. The outer cortex is supplied by the periosteal vessels. Lack of adequate muscular covering, as in the tibia or clavicle, decreases this periosteal blood supply substantially.

How many times is a tibial fracture seen in which a closed reduction with a slight offset would have been better than the open procedure which was done to obtain the anatomic reduction that is so eye appealing on radiographic examination? In metaphyseal areas where there is an abundant blood supply and good osteogenetic tissue, an even greater offset may be permitted than in the diaphyseal areas. Too often it is forgotten that such edicts as Wolff's law do exist and do work.

The works of many brilliant men have established the fact that bone is not a dead skeletal structural framework such as the steel supports of buildings, but rather a living cellular tissue consisting of an organic matrix impregnated with inorganic substances. This organic matrix consists of a collagenous protein, ossein. The inorganic substances consist chiefly of calcium phosphate with the addition of magnesium phosphate and traces of other substances such as fluorine.

Osteoblasts arise from the mesenchymal cells and remain active through life as a layer of cells deep in or just under the periosteum and in the endosteum. They develop into osteocytes and as such become part of the bone itself. They lie in and completely fill the bone cell cavities, or lacunae. Osteoclasts are also found in bone and are believed to be responsible for bone destruction. There is some controversy as to their origin; however, it is generally conceded that they arise either directly from the mesenchyme or by fusion of osteoblasts.

A group of enzymes has been discovered which is somehow associated with bone formation. These enzymes bring about the liberation of inorganic phosphatase through hydrolysis of organic phosphorus compounds. It is probable that many such enzymes exist, but their activity, as in the case of all enzymes, depends upon the hydrogen ion concentration of the surrounding fluids. That nature changes this hydrogen ion concentration in fractures goes without saying; however, I wonder how much more radically it is upset when open reductions are done.

The phosphatases can be divided into two main groups, alkaline and acid. The alkaline group is by far the more widely distributed in the tissues of the body. Acid phosphatase is mainly concentrated in the prostate gland, and apparently arises in the epithelium of this gland. It is also present to some extent in the spleen, the brain, and possibly the liver. Phosphatase is present in large amounts wherever active calcification takes place, and it is believed to be manufactured by the osteoblasts. The alkaline phosphatase is also found in the kidney where it appears to influence the resorption of glucose, in the liver, and in white blood cells. It is excreted in the bowel, and so the serum level may be increased in obstructive jaundice. Most authors believe that this enzyme alkaline phosphatase plays a special part in bone formation.

HEALING OF FRACTURES

With the disruption of vessels and bone following fracture, a hematoma is formed immediately. This is spindle shaped on each side of the fracture, both in the medullary part and under the periosteum. If the periosteum has been torn, the hemorrhage extends into the soft tissues. The surrounding tissue becomes hyperemic and edematous with or without tearing of the periosteum. The hemorrhage and dead tissues found at the fracture site soon provoke an inflammatory reaction which passes over the repair state. Within 48 hours after injury, organization of the hematoma, formation of blood vessels, and the appearance of fibroblasts are already in evidence. At the same time, mitotic figures appear in the deep layers of the periosteum and endosteal cells. These cells proliferate, lifting up the fibrous periosteum and producing a fusiform enlargement on each side of the fracture. If all goes well, the proliferation continues and folds over into the break, finally causing continuity of bone and thus establishing bony union. After the two broken ends have been united, remodeling and consolidation occur. The cartilage is absorbed and bone is laid down. Finally, the irregular bone is replaced by lamellar trabeculae which follow the lines of stress and strain (Wolff's law). The periosteal callus is absorbed, and after a time the repair is so complete as to give the normal appearance of bone.

This healing occurs in three stages: The first stage is that of repair with granulation tissue. This is the essential process in the union of any fracture and forms the scaffolding for the second stage which is union by primary callus. The calcium cells are deposited in the granulation tissue around and between the bone ends. The final stage is that of true bone formation and perfect union. In the formation of callus, three connective tissues are represented: fibroblastic, chondroid, and osseous.

Repair of a fracture is primarily a local process and will occur even when the general condition of the patient is quite poor. Healing will occur even though the calcific loss has been so great that the fracture was spontaneous. In severe osteoporosis, rickets, and gastrocolic fistula, where calcium and phosphorus absorption is minimal, it is interesting to note that sulfanilamide has a depressing action upon phosphatase activity.

Burns and Young[2] in 1942 stated that about 1930 deterioration in the rate of union of fractures suddenly became apparent. It was then that plaster casts became popular and almost completely replaced wooden splints, thus leading to more complete immobilization. These same authors claim that a certain amount of movement favors callus formation and that early weight bearing or compression at the fracture site is beneficial. McMurray[3] in 1942 also stated that the early use of plaster delays union by preventing the formation of hematoma to form the scaffolding for callus formation. On the other hand, Watson-Jones and Coltart[4] hold that fractures unite as quickly today as in the past and that factors causing delay are interrupted immobilization, traction, distraction, infection, disturbed blood supply, and too early weight bearing. If anatomic, physiologic, and pathologic knowledge of the fracture site is utilized, it is easily seen that plaster applied too tightly, which would interfere with a hematoma and local blood supply, as well as imperfect immobilization, interferes with the granulation of callous tissues.

Utilization of closed reduction requires as much skill and judgment as any open reduction, and, in my opinion, it requires considerably more. I sincerely believe that any orthopedist could teach a student or a graduate nurse the mechanical technics of open reduction in a few short lessons, but few could impart that degree of judgment necessary to produce good union. Unfortunately, too many of us have not learned it ourselves.

COMPLICATIONS OF FRACTURE HEALING

Six complications of fractures that should be mentioned are: (1) delayed union and nonunion, (2) vascular complications, (3) nerve complications, (4) soft tissue ossification and/or fibrosis, (5) avascular bone necrosis, (6) infection-osteomyelitis.

Delayed Union and Nonunion.—

Etiologic factors of delayed union and nonunion include:

1. Inadequate reduction
2. Overseparation of the fractured parts (This is commonly seen when skeletal traction is utilized. Too large a gap remains to be bridged with adequate callus. It is also frequently seen in the utilization of a Lane plate that does not allow normal impaction to occur as healing progresses and nature cleans out the debris at the fracture site. This has been overcome, at least in part, by utilization of Egger's slotted plate.)
3. Fracture of two bones of an extremity where one bone heals faster than the other and does not allow the normal impaction to occur
4. Interposition of soft tissue
5. Excessive radiation
6. Inadequate immobilization (This might well be due to plaster that is applied too tightly or too soon, before swelling has occurred, or inadequate external fixation of the plaster allowing motion to occur at the fracture site.)
7. Extensive damage to soft tissue (Often upon opening a fracture of the lower tibia and in cutting through the skin and superficial fascia, this area is found to be as edematous as if the area had previously been infiltrated with excessive amounts of procaine; a short time later the additional trauma is found to have caused more damage which results in an aseptic slough of the wound.)

Vascular Complications.—Beyond a doubt some arterial spasm is associated with all fractures. Nature's protective mechanisms sometimes become overactive. Realization of the importance of arterial spasms has led to a revision of views of the essential pathology of conditions such as Volkmann's ischemic contracture. It was formerly believed to be due to venous obstruction caused by plaster that was too tight. It is now believed to be brought about by overactivity of the protective mechanisms, with too severe and prolonged arterial spasm. Sudeck's atrophy, which with its prolonged disability fortunately occurs but rarely, is more evidence of this disturbance of arterial contraction.

Infection.—

By briefly discussing some history, anatomy, and pathology of fractures I have attempted to show that certain complications are more frequent with open reductions than with closed reductions. Of all the complicating factors of fractures, especially of open reduction, infection is the most important. It is a low-grade infection that is different from the acute hematogenous osteomyelitis that is adequately controlled today as

compared to yesterday. In my opinion, the chronic osteomyelitis occurring after open reduction remains an unsolved problem, in spite of newer antibiotics, sensitivity tests on the organism, improved surgical technics, and so forth. The addition of metal, provoking unnecessary cicatrix with a knife, installation of sulfa into the wound, destruction of vessels by section and ligatures, and associated neurotrophic changes due to local trauma to vessel-controlling nerves not only contribute to nonunion, but lay the cornerstone for infection by tearing down the body's most powerful defenses.

Starr,[5] in his thought-provoking monograph on delayed union and nonunion, states, "If the dread of bone infection following operation on fractures remains, like the sword of Damocles, over our therapeutic head during the next decade, surgical training, not the pathology of bone, is at fault." I have no quarrel with that statement. When indications exist for open reduction, as when adequate closed reduction cannot be obtained, no thinking physician would hesitate. Delaying operative intervention in fractures of the hip in the aged is the prelude to signing the death certificate.

It is not with the indications for operative intervention that I quarrel, but rather with the interpretations of these indications. Too many in private practice, who do not spend time in large charity hospitals, fail to realize the morbidity and great economic hardships created by infections following open reductions. I sincerely believe that all surgeons tend to close their eyes to their bad results. It would be better if these cases in which infection has occurred were followed up with clinical and radiographic examinations at 6-month or yearly intervals, just as cases of cancer are followed up. With this plan it would be comparatively simple to discover an early Brodie's abscess or an early sequestrum and remove it before it is beyond control.

After operative reduction if infection sets in, the patient all too frequently finds that he is unable to bear the financial burden it creates. In pseudogenerosity, he is sent to a public institution where follow months or years of prolonged disability and drainage, repeated surgery which all too often is to no avail, and finally amputation. Amputation, in this case, is the surgeon's admission of defeat.

When this patient is the breadwinner of the family, economic problems arise that rarely can be coped with by the average working person, and both the patient and his family suffer. Dr. Morhardt's[6] well-known statement, "Every oophorectomy should be considered by the surgeon as a personal orchidectomy," might well be paraphrased to apply to a painful and infected fracture following operation.

In August of this year, I performed a mid-thigh amputation on a 45-year-old man whose right tibia became infected in 1934. A compound fracture had occurred as the result of an oil-field explosion. He was in the field about an hour before he reached a hospital. Following hospitalization and debridement he was kept in traction for about 18 days. Then Steinmann pins were placed for adequate alignment, and a plaster cast was applied incorporating the pins. His leg was in a cast for the next 2 years, during which time the injury continued to drain and frequent cast changes were necessary. The wound was approximated with adhesive to obtain adequate soft tissue bridging.

Following removal of the plaster he was placed in a brace for 4 years; after that length of time the brace was worn out and was discarded. In 1946 a small piece of bone worked itself out of the wound, and the wound drained for a week. There was no drainage after that, and the patient remained asymptomatic until December, 1950, when some pain, swelling, and a small sinus developed. At that time he was admitted to the Los Angeles County Osteopathic Hospital. In June, 1951, a sequestrectomy and curettage were done. In July, 1952, a saucerization and another curettage were performed. In October, 1952, a left thigh tube graft was started, which died when placed at the recipient site in November, 1952. In December, 1952, saucerization was again performed and an abdominal split-thickness graft was unsuccessfully attempted. In July, 1953, an attempt was made to drift some skin over the extensively saucerized tibia and once again, skin slough occurred. All these measures were taken and, in spite of all the laboratory tests, consultations, antibiotic therapy, radiation, et cetera, available in the Los Angeles County Hospital, complications still developed. There is no need to dwell further on such a case history. This case was described only to illustrate what can happen when infection occurs in the treatment of fractures—after either closed or open reduction. The pain, disability, and economic loss to the individual and to society cannot be estimated.

It is possible that increase in hospitalization programs and eagerness to cut down time away from work may have influenced the surgeon's judgment in favor of the open reduction. However, keeping the picture of this man in my own mind, and placing myself in his position, I find it exceedingly difficult to perform open reduction when the indications are debatable. In addition, having had osteomyelitis and knowing the morbidity, pain, repeated surgeries, prolonged disability, and economic hardships it imposes, I can only say that if all surgeons would keep this picture of infection in mind when they pick up the scalpel, they would perhaps try a little harder to obtain adequate closed reduction.

SUMMARY AND CONCLUSIONS

1. An attempt has been made to stimulate objective rereading of the history of orthopedics.

2. A brief review is given of the salient facts of blood supply and composition of bones as they pertain to fractures.

3. It is suggested that periodic checks be made in infected cases similar to follow-up examinations in cases of carcinoma.

4. It is my opinion that the indications for open reductions of fractures are too freely interpreted.

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Fractures of the Mandible

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The modern orthopedic and traumatic surgeon is frequently confronted with fractures of the mandible and other facial bones. Often these fractures are accompanied by other bodily injuries, usually as a result of automobile accidents. Treatment of these conditions is an important branch of traumatic surgery and one that is poorly understood by the average surgeon.

The anatomic features of the jawbones differ from those of the other bones of the body, and because of the unusual construction and function of the jaw, special methods of surgical management are required in the repair of these fractures. The teeth create an unusual situation, and much consideration must be given to them. For example, teeth that are in the fracture site must be extracted and the fracture then becomes compound and must be treated as such. An irregular appearance of the teeth following injury is suggestive of a fracture, and the restoration of regular appearance is suggestive of proper alignment. In conservative treatment, the teeth may serve as a natural means of anchorage in reduction and fixation of the fracture. Many times the treatment of choice is open reduction.

Many methods have been devised to control fractures of the mandible, but all are based on three fundamental methods of fixation, namely, wiring, splinting, and elastic traction. There are many modifications of these three classifications which vary from ultraconservative to very radical open reduction procedures.

The importance of early treatment with accurate reduction and control of infection cannot be overemphasized. However, accompanying cerebral concussion, shock, severe hemorrhage, and other severe injuries may prevent early care. As a rule, antibiotic therapy can be initiated immediately, and local treatment of lacerations and establishment of adequate drainage can usually be carried out without injury or adding to the complications of other injuries.

ETIOLOGY AND CLASSIFICATION

In civil life the most common source of injury is a blow as a result of a fist fight. The next most frequent causes are automobile accidents, falls, kicks, and blows from various missiles.

Classification of fractures of the mandible usually is the same as for other fractures, with the addition of an extra classification, the depressor or elevation type, which is a classification in respect to the displacement by muscle pull. Under normal conditions, the depressor and elevator muscles, acting in groups, have a reciprocal action in carrying out the functions of the jaw. The depressor group of muscles is composed of the mylohyoid, the digastric, and the geniohyoid, all of which have their attachments on the inner surface of the body of the mandible and pass inward and downward and backward to the hyoid bone. The elevator group is composed of the masseter, the temporal, and the internal pterygoid muscles. This group results in powerful upward and backward and outward pull on the ramus. These are the groups of muscles responsible for the marked displacement of fractures of the mandible, and their origin and insertion and action must be taken into consideration in the treatment of fractures.

The highest percentage of fractures occurs at the angle of the mandible. Next in frequency are those at the cuspid region, the bicuspid and molar region, the neck of the condyle, the symphysis, the ramus, and the coronoid process.

EXAMINATION AND DIAGNOSIS

The objective symptoms will be the determining factors in arriving at a positive diagnosis. They include swelling, discoloration, deformity, loss of function, false motion, crepitus, and irregularity in the tooth line. X-ray examinations are not only valuable in diagnosis but are essential in selecting the proper means of fixation and reduction.

The surgical team should consist of the traumatic surgeon, his immediate assistant, a dental surgeon, and a qualified anesthetist. The anesthetist is a very important member of the surgical team, particularly in guarding against the seepage of blood and secretions from the mouth which is so common in these injuries. In my opinion intratracheal anesthesia is the anesthesia of choice.

IMMOBILIZATION

Many authorities on fractures of the jaw advocate the type of immobilization which permits slight movement of the fracture site and supposedly aids materially in repair. They believe that a fracture which is held in perfect anatomic position will not repair as rapidly as one that over-rides slightly and is not completely immobilized. I do not agree with this argument, and in my opinion it lacks the endorsement of clinical research carried out on a modern and scientific basis. Some general surgeons think there is an advantage to a slight amount of motion at the site of fracture, but again I believe that the accumulated clinical experience of orthopedic surgeons and extensive research on bone regeneration and repair indicate otherwise. Every effort is made by the orthopedic surgeon to immobilize securely the fractured surfaces of bone in contact with each other. This is an established principle of fracture surgery of long-proved value, and one should hesitate to make any departure from it. I strongly suggest a firm method of immobilization with complete absence of motion if possible, in order to restore perfect alignment of the mandible.

TECHNIC

I feel that the osteopathic concept of disease is very applicable to fractures of the mandible since much attention is given to improving the circulation and nutrition of the tissues and muscles in the region of the fracture by means of osteopathic manipulative therapy, physiotherapy, and early movement of the fractured parts. The method of immobilization that will be described is based on these principles; it allows immobilization of the fracture site and movement of the related parts. I seldom use the technic of wiring the teeth as the method of fixation or elastic bands for fixation and traction, because with these technics some motion seems to exist at the fracture site.

My choice for immobilization of mandibular fractures is the Tower splint apparatus. This fixation apparatus has setscrews securing the Tower screws that enter on each side of the fracture site, protrude through the skin, and secure to a metal bar which serves as a splint. These bars come in a variety of sizes, some straight and some curved for the curvature of the mandible. The surgical approach is made directly over the fracture site for 2 to 3 cm., depending upon the obliquity of the fracture. After the fracture is reduced the pins are placed either above or below the incisional line, in a sort of blind-pinning technic, usually far enough from the incisional area to allow a plastic closure, thus resulting in a minimum of scar tissue. Usually two to three pins are required in each fragment. A small shield is provided to fit over the bar, with adequate surgical dressings over the incisional area. It is seldom necessary to immobilize the temporomandibular joint when this method is used, and the patient can immediately take food without difficulty. Of course, heavy chewing of solid food is avoided until time has elapsed for complete healing.

AFTER-CARE AND COMPLICATIONS

After-care is greatly simplified by this method and consists chiefly of preventing infection. I routinely use penicillin and streptomycin in these cases until the fifth to sixth postoperative day. Sulfathiazole chewing gym is also used routinely, both for its antibiotic effect and because it exercises the soft tissues. The screws are removed in 6 to 8 weeks. They can be taken out without an anesthetic and with little pain to the patient. A small bandage is placed over the pin opening, which usually heals in 2 or 3 days.

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Intramedullary Pinning of the Clavicle

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Intramedullary fixation, which has proved highly successful with various fractures of long bones, is now becoming highly favored for fractures of the clavicle. Other operative methods of fixation of clavicular fractures have been highly unsatisfactory. External skeletal fixation devices did not adequately immobilize the fracture site, were difficult to apply, and frequently produced excessive scarring. The use of bone plates was not practical. Osteosynthesis with wiring of the fractured ends was unsatisfactory because of inadequate immobilization and breaking of the wire. Recently, intramedullary fixation has been used by the authors and numerous other surgeons. It has been found that this method eliminates practically all of the objections to other methods. It has been so satisfactory that open reduction is more and more frequently being considered the procedure of choice in any displaced fracture of the clavicle.

Several methods of intramedullary fixation of the clavicle have been used, including threaded pins, small Steinmann pins, and various sizes of Kirschner wires. Moore[1] reports the use of 3/32 inch Steinmann pins on 25 cases, all with good results. McKeever[2] advocates the use of 1/8 inch threaded pins. Geckeler[3] reports 47 good results in 48 cases with the use of various sizes of Kirschner wires, depending upon the size of the bone. Ripstein[4] lists several indications for Kirschner wire fixation of the clavicle. These include:

1. Oblique fractures of the middle third of the clavicle with displacement
2. Almost all fractures of the outer third of the clavicle
3. Cases in which a good cosmetic result is desired
4. Cases in which immobilization of the arm or shoulder would impose a severe handicap on the patient or on the care of the patient.

To these may be added any displaced fracture of the clavicle, especially in the presence of comminution. The method would seem to be ideal for fixation of osteotomies of the clavicle incidental to other surgery.

There are no specific contraindications to Kirschner wire fixation of the clavicle, except for fractures of the sternal end, in which the method is unsatisfactory.

Much wider use of intramedullary pinning of the clavicle is advocated for the following reasons:

1. Ease of operation
2. Short duration of surgery (15 to 40 minutes)
3. Ease of fixation with additional immobilization seldom required
4. Comfort of the patient
5. Use on any age group (this series 4 to 88 years)
6. Good cosmetic results with generally very little scar
7. Rapid healing time as compared with other methods of fixation of displaced fractures; healing comparable to that in nondisplaced fractures
8. Good functional results.

Fig. 1

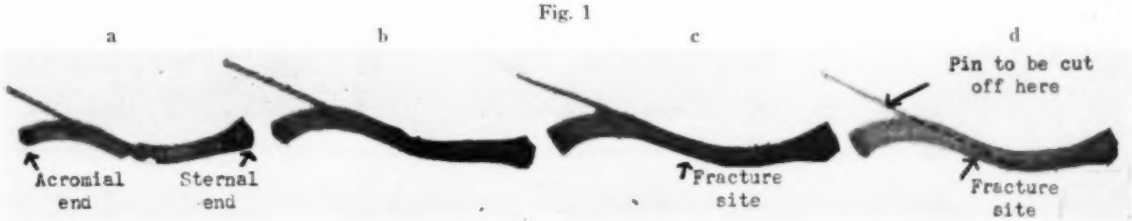


Fig. 2

The technic of the procedure is as follows:

A local anesthetic may be used if a general anesthetic is contraindicated.

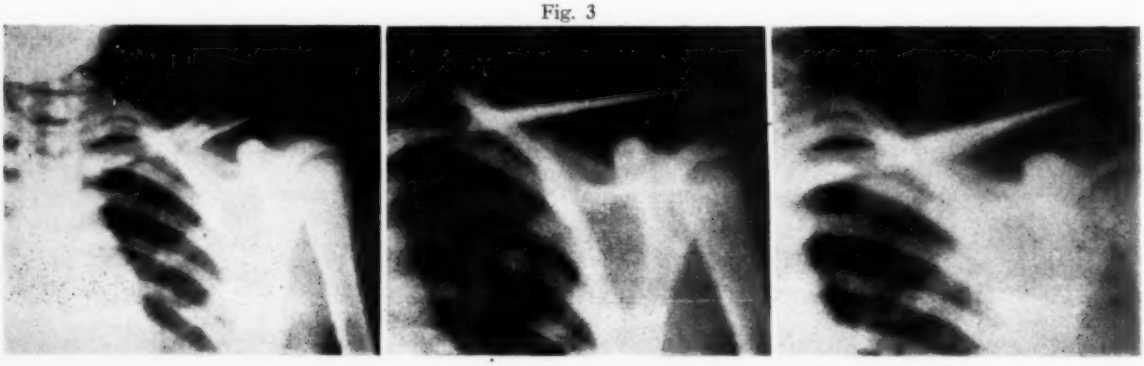
The patient is supine with the involved shoulder elevated on a sandbag or pillow. Incision is made directly over the fracture site and carried down to expose the fracture. Any interposition of tissue is cleaned out, and small loose fragments are removed. The distal fragment is held with a bone grasper while a drill point (the size of the Kirschner wire to be used) is inserted into the medullary canal with the drill through the posterior outer cortex of the clavicle (Fig. 1a). The Kirschner wire selected will range in size from 1/32 to 3/32 inch in diameter. It is recommended that both ends of the wire be sharp. The wire is inserted with the drill into the medullary canal and out the posterior cortex of the clavicle through a stab wound in the skin on the posterior superior aspect of the shoulder. It is inserted until the proximal end is flush with the fracture site (Fig. 1b). The fracture is then reduced (Fig. 1c) and held in place with bone graspers. Then, either with a drill or a mallet, the wire is inserted approximately ½ to 1½ inches into the medullary canal of the proximal fragment (Fig. 1d). This pro-

TABLE I—CHART OF CASES TREATED BY INTRAMEDULLARY PINNING METHOD*

No.	Sex	Age	Date Injured	Date of Surgery	Date of Hospital Discharge	First Recheck	Second Recheck	Third Recheck	Date Pin Removed	Date Discharged	Weeks of Healing	Final Result	Complications	Other Injuries
1	F	47	11-22-51	12-10-51	12-31-51	12-31-51	2- 7-52	4- 6-52	4- 6-52	4-17-52	9	Fair	Stiff shoulder	Fractured scapula and ribs
2	M	13	3-18-52	3-18-52	3-21-52	4-18-52	5- 9-52		5- 9-52	5- 9-52	7	Good	None	
3	M	7	5- 1-52& 5-13-52	5-14-52	5-16-52	6- 4-52	6-16-52		6-16-52	6-16-52	5	Good	None	
4	F	26	6-30-52	7- 1-52	7- 3-52	8- 4-52	8-25-52		8-25-52	8-25-52	8	Good	Some displacement	
5	F	35	8-21-52	8-25-52	8-27-52	9-18-52	10- 9-52	10-31-52	10-31-52	10-31-52	10	Good	None	Concussion
6	M	15	9- 4-52	9- 4-52	9- 5-52	10-13-52			10-13-52	10-13-52	5	Good	None	
7	F	88	9-16-52	9-17-52	9-19-52	11-11-52			11-11-52	11-11-52	8	Good	None	
8	M	14	10- 2-52	10- 3-52	10- 5-52	11-14-52			11-14-52	11-14-52	6	Good	None	
9	M	5	11- 7-52	11- 8-52	11-13-52	12- 5-52	12-20-52		12-20-52	12-20-52	6	Good	None	Concussion
10	F	58	11-27-52	11-28-52	12- 2-52	2- 1-53			2- 1-53	2- 1-53	8	Good	None	
11	M	6	12-16-52	12-16-52	12-18-52	12-25-52	1-27-53	2- 8-53	2-28-53	3-30-53	11	Good	Drainage pin site Granulation	Concussion
12	M	11	12-15-52	12-16-52	12-18-52	1-24-53			1-24-53	2- 1-53	5	Good	None	Concussion
13	M	9	1-11-53	1-12-53	1-14-53	2- 7-53	2-21-53		2-21-53	2-28-53	6	Good	Stitch abscess	
14	F	58	7-11-53	7-11-53	8- 5-53	Died						Died	Pulmonary thrombus and embolus	Fractured pelvis
15	M	4	8-15-53	8-17-53	8-19-53	9-22-53	10- 6-53	10-27-53	10-27-53	10-27-53	9	Good	None	
16	F	68	8-19-53	8-20-53	8-24-53	10-16-53			10-16-53	10-16-53	8	Good	None	
17	M	69	8-22-53	8-24-53	9- 3-53	9- 8-53	10- 9-53	11- 2-53	9- 8-53	11- 2-53	10	Poor	Sternal end fracture displaced and pin migrated	Previous thoracoplasty
18	M	16	9- 3-53	9- 4-53	9- 8-53	10-13-53	11- 6-53		11- 6-53	11- 6-53	9	Good	None	
19	M	5	9-12-53	9-12-53	9-15-53	10- 3-53	10-14-53		10-14-53	10-14-53	5	Good	None	

*These cases were treated in the period from December, 1951, to September, 1953. Thirty-four cases were treated by the closed method during the same period.

Fig. 3



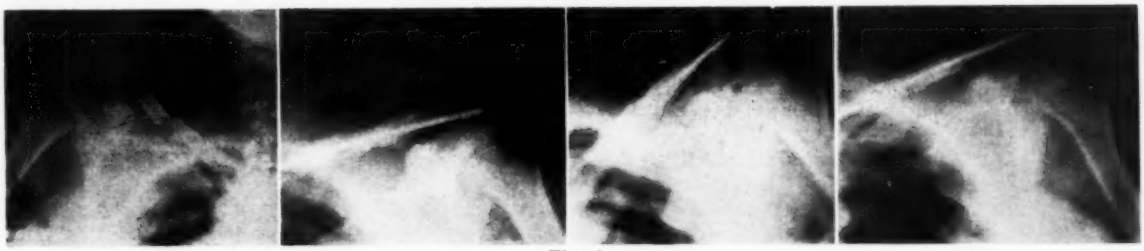


Fig. 4

vides adequate and sure fixation. The end of the wire protruding through the skin is then cut off under the skin edge. Sufficient length protrudes from the bone to permit later removal. The wound is closed in layers in the usual manner and sterile dressings are applied.

The arm is taped across the chest for the first 24 hours and then the patient is permitted to wear a double sling or a Velpeau bandage. The normal triangular sling is used with a folded triangular sling wrapped around the arm and chest to prevent swinging of the arm. By the time the patient leaves the hospital he generally needs only the usual triangular sling. Sutures are removed on the tenth postoperative day. Generally, the wire is removed under local anesthesia through a small stab wound as soon as adequate healing is indicated by x-rays. This occurs from the sixth to the tenth week after operation.

The accompanying chart presents a review of 19 cases with 16 good results, 1 fair result, 1 poor result, and 1 death. The death was due to complications from other injuries. The poor result was a sternal end fracture. In this case the wire became loose and pulled through the sternum where it was inserted, permitting redisplacement. Other complications in this series included a moderately stiff shoulder, some drainage from the pin site with excess granulation tissue formation, stitch abscess, and partial recurrence of displacement in a fracture of the outer third. Migration of the pin occasionally occurs but only distally. The average healing time was 7½ weeks. The average time under care was about 9 weeks. The age range was from 4 to 88 years. During this period (approximately 18 months) 34 clavicular fractures were cared for by closed methods.

X-rays of three typical cases in the series (Figs. 2-4) are presented showing the original fracture, the immediate postoperative result, and the final film prior to pin removal.

SUMMARY

Intramedullary fixation of fractures of the clavicle is frequently indicated. The method is characterized by ease of operation, ease of fixation, comfort of the patient, and good cosmetic result. The technic has been given. A review is made of 19 cases, giving almost uniformly good results, with the healing time comparing satisfactorily with that of undisplaced fractures of the clavicle. Further use of this method is recommended.

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THE NEED FOR BASIC INVESTIGATION IN ORTHOPAEDIC SURGERY

Orthopaedic surgery today is lacking in basic research, and too little animal experimentation is being done. The only gain in knowledge in the field of surgery is through observation, experimentation, and inductive and deductive reasoning. No sound progress is to be expected from the Aristotelian principles of inspiration, intuition, and dogmatism. In the broad field of surgery during the past twenty-five years there has been tremendous progress, and this progress has been due to fundamental investigation in physiology and biochemistry. Many conditions which fifteen years ago were treated on a purely empirical basis are now approached by sound scientific methods because through basic investigation they are more clearly understood. Although the orthopaedic surgeon during the past quarter century has been quick to apply the results of all basic investigation and to incorporate these results in his armamentarium for the clinical management of patients, he has not himself, I believe, contributed enough of the blood, sweat, and tears which go into the advancement of general surgical knowledge as it applies both to the broad field of surgery and to his own specialty.

To make this point more concrete, I might cite some of the advances of the past quarter century in the general field of surgery. The management of shock and the problem of fluid replacement have moved out of the empirical stage. These are both problems with which orthopaedic surgeons are associated daily. A perusal of the basic research in these subjects does not reveal the names of many people trained in orthopaedic surgery. Again, in the conquest of the field of infection none of the basic work in chemotherapy and antibiotic therapy can be claimed for our specialty.—Francis M. McKeever, M.D., *Journal of Bone and Joint Surgery*, April, 1953.

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Diagnosis and Indications for Surgical Management of Ruptured Lower Lumbar Disk

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It has been 98 years since Virchow[1] described the intervertebral disk and 57 years since Kocher[1] reported rupture due to acute trauma, but it has only been during the last 20 years that the great volume of work has been done in definitely proving the ruptured disk as a distinct clinical entity. Since the original work of Mixter and Barr[2] and others, many thousands of cases have been studied and volumes have been written, until the ruptured disk syndrome is now rather well understood by most students of spinal pathology.

Variants will always be noted to prove that the syndrome is not constant, and the experiences of individual surgeons in this particular field will add to the sum total of knowledge to prove once again that, while cases may be similar, no two individuals are exactly alike.

Disks rupture at points of unusual wear and tear or points of great mobility, notably at the lower cervical portion of the spine and also in the lower lumbar area. Disks will rupture in the upper lumbar, and also occasionally in the dorsal spine, but without the degree of frequency that warrants more than a mention in this paper. The lumbosacral disk ruptures in approximately the proportion of two to one in relationship to the disk above.

It might be well to review briefly the anatomy of the intervertebral disk and to remember that these cartilaginous cushions serve as a shock-absorbing mechanism from the second cervical to the lumbosacral portion of the spine. The nucleus is enclosed by circular fibers known as the annulus fibrosus, and the nucleus is also confined within its normal position by the upper and lower cartilaginous plates of the bodies of the two adjacent vertebrae. Since the nucleus pulposus is the troublemaker in these cases, it is well to remember that this nucleus is also maintained within its normal confines by the anterior and posterior longitudinal ligaments. The adjacent ligamenta flava also act to maintain facet stability and, as a consequence, to maintain the nucleus within its normal confines. The disk will bulge in all directions under pressure and will return to normal when the force is released.

Inman and Saunders[3] have analyzed the leverage involved when the lumbar spine is in flexion, and their reports strongly support the belief that the original force applied to the flexed spine need not be great in order to exert a tremendous pressure on the disk. Knowledge plus clinical application of those findings has been extremely useful in eliciting a diagnostic history from the patient with a ruptured lower lumbar disk. It is exceedingly common to find that one of the causative factors has been either a severe injury to the lower lumbar spine produced by heavy lifting while in the stooped position, or repeated minor injuries to the lower back as a result of occupational strains and stresses while the individual is in the flexed position. Degenerative changes in the disk which have been produced over a period of years by neurocirculatory changes act as contributing factors to an ultimate rupture, and congenital anomalous changes in the lower lumbar spine with congenital changes in the disk itself also eventually tend to manifest themselves in a frank rupture of the disk.

Therefore, in seeking to evaluate the causative factors of disk ruptures, it is necessary to consider postural changes over a period of years, congenital defects in the bony structure as well as in the disk itself, repeated minor traumata over a long period, and sudden acute injury which may rupture the disk quickly and forcefully. Postural changes are those changes which take place because of posture which is considered to be improper, that is, in the slumping child, in the youngster who stands persistently on one foot while at rest, in the child who sits on one leg, and in the extremely lordotic youngster who places a more or less constant strain upon the lumbosacral facets because of the increased angulation of the sacrum. Such bad postural habits carried into adulthood certainly will act to produce an excessive amount of wear and tear upon the lumbosacral disk structure, and, as a consequence, can be some of the very direct causative factors behind eventual rupture at that point.

Congenital or acquired short leg can produce this same effect by producing a definite tilt of the pelvis, which in turn places an undue strain on the disk structure of the lower lumbar spine. Certainly if good postural prophylaxis is to be taught it should begin early in childhood. Undoubtedly, then, many of the disk ruptures seen in later life could be prevented if such measures were taken early in the individual's life.

Developmental defects such as unilateral sacralization, spina bifida occulta, and other developmental changes in the lower lumbar spine also act to produce secondary strain upon a disk which may also be involved with a congenital defect in the annulus, and, as a consequence, it would seem that developmental congenital defects could be considered etiologic for this condition. Many times at surgery actual frank tears are found in the interspinous and supraspinous ligaments which secondarily tend to produce joint instability with a greater strain then thrown upon the supporting structures and a greater strain also thrown upon the nucleus and annular fibers themselves. These tears may well represent the end result of a congenital developmental defect in the ligament and/or may be entirely due to some previous trauma applied directly to that portion of the spine.

It is recognized that instability in the vertebral articulation would be more apt to occur as a result of this weakening effect upon the adjacent ligaments, and, as a consequence, would have a tendency over a period of years eventually to break down a weakened annulus with ultimate extrusion of the nucleus. The actual plane of the posterior articulations has an important effect upon intervertebral movement. In the dorsal region, the planes of the articulation lie in the axis

of rotation of the vertebral bodies, one on the other, while in the lumbar and cervical regions, the plane of the articulations lies across this axis and prevents rotation in these regions. In the lumbar region, the articular facets, with the exception of those in the lumbosacral joint, lie more or less in the sagittal plane. The lumbosacral articular facets lie more in the coronal plane. There are, however, many variations of this arrangement, and the direction of the facets is often irregular and may even differ on the two sides.

These congenital anomalous changes often act as predisposing factors in determining the degree of instability which goes into the production of an ultimate disk rupture. Actual subluxations of the lumbosacral articular facets, whether of an anomalous nature or in the position considered normal, certainly would act as one of the chief predisposing factors, productive of changes of a neurocirculatory nature which could well lead to eventual disk rupture. I refer to those early subluxative changes in a joint due to minor traumata during childhood. These changes go unrecognized at the time but lead to degenerative changes in the disk structure itself and are discovered in adulthood only at the time of surgery. These patients undoubtedly represent that class of individual who has no distinct remembrance of having any injury to his back which would predispose to the condition which brings him for medical attention. There are many patients who have no distinct remembrance of any injury, but who probably could be classified in that group whose early subluxative changes in childhood are now manifesting themselves as actual disk ruptures in adulthood.

The individual who has maintained articular lesions in the lower lumbar segments of his spine over many years, with added occupational stress during his adult years, is the one who is predisposed to disk damage with eventual rupture. This rupture may not come until after 20 to 25 years of adult life, but it may also come during the teen-age period if occupational forces applied to the lower back are more than the person is constitutionally fitted for. I have operated on and removed a ruptured disk from a 16-year-old boy and also from a 67-year-old man. In the first instance the patient had three ruptured disks, brought on by acute trauma, which were removed at surgery. The boy had performed exceedingly heavy work from the age of 10 with a body not functionally adequate for such arduous occupation. The 67-year-old man was an individual who had compensated well for over 30 years before the last lumbar disk broke down and produced direct nerve root pressure.

It has been said that sciatic pain of a rather distinct radicular nature, with marked tenderness over the spinous processes of the last two lumbar vertebrae, can be considered to be nerve root pain due to a disk rupture until proved otherwise. This, of course, does not definitely place all cases of sciatica in the field of the ruptured disk, but at least it places the great majority in that field, and, ruling out caudal or nerve root tumors, practically all cases of a sciatic neuralgia are found to have a ruptured disk as the etiologic agent.

Almost every physician has seen cases of acute prostatitis which also produced associated sciatic neuralgia. Probably all general surgeons of much experience have had the satisfaction of removing tumors from the female pelvis and relieving low-back and associated sciatic pain. On the other hand, it is entirely probable that lumbosacral facet disease, sacroiliac subluxations, and other vertebral lesions of the lower lumbar spine can influence and perhaps produce some degree of sciatic pain. Certainly osteopathic physicians have been influencing the course of this disease by manipulative therapy over many, many years, and it is not entirely illogical to believe that there are those cases where a sciatic distribution of pain is based entirely on subluxations of the articular facets. It is also known that pain associated with true disk rupture has been influenced by osteopathic manipulative procedures, and, as a consequence, it is many times difficult to tell without surgical investigation whether or not a true disk rupture has been present or whether the sciatic pain has been entirely based on the so-called facet syndrome.

Very often definite arthritic changes in the lumbosacral facets are seen which in themselves can act as a positive factor behind the production of pain, especially in the presence of a degenerative disk disease or osteoarthrosis, with some consequent narrowing of the intervertebral foramina. While I believe that the arthritic changes present are secondary to an unstable disk, nevertheless, after the cartilaginous surfaces of the facets have undergone pathologic change, it is not difficult to understand why this factor then also becomes one of the cogs in the mechanism of pain production. It is usually in this case that pathologic or hypertrophic changes are also found in the ligamenta flava. It must be remembered that the ligamenta flava form the posterior boundary of the intervertebral foramina, and, as a consequence, hypertrophic changes, especially in the subflavum ligaments, can also influence changes in the nerve root as it passes through the foramina.

In old, long-standing cases of low-back pathology with eventual rupture some degree of hypertrophy of these ligamenta is seen and, not infrequently, adhesive changes between the ligament and the dura itself. The ligament can also act to compress a nerve root when the subflavum or inferior portion of the ligament becomes thickened and, with the root displaced posteriorly, owing to a retropulsed disk, the mechanism of compression becomes quite obvious, and the secondary role played by the ligamentum flavum is also obvious. I have felt, however, that the ligamentum flavum acting as a compressing agent, without a true disk rupture, is not a common phenomenon. I feel that usually both a ruptured disk and the associated thickening of the ligamentum flavum are required to produce compressive changes against the root proper. Perhaps the only exception to this would be the congenitally defective disk which has ended up as a true osteoarthrosis, or a frank intervertebral narrowing without posterior extrusion. In this type of case, the effect of the thickened ligamentum flavum upon the narrowed foraminal opening is seen, and actual nerve root changes do occur as the root passes through the foramen.

Sciatic pain, as the chief and predominant symptom of disk rupture, is usually of a unilateral nature. Patients generally give a history of repeated attacks of so-called lumbago or lower lumbar instability with muscle spasm but without an associated sciatic pain over a period of years. Eventually an attack is complicated by distinct and definite unilateral sciatica. Then patients state that on previous occasions their low-back pain soon cleared up, but on this occasion it has been irresponsive to treatment. It is now this sciatic involvement which brings them to the specialist.

In evaluating suspected lower lumbar disk rupture it is absolutely necessary that a rather complete and detailed routine examination be done on all patients complaining of low-back pain, especially the patient complaining of low-back pain with associated sciatica. Both positive and negative findings should be recorded on the examination as complete clinical records are necessary to evaluate properly the patient's chief complaint and detailed progress if conservative or other treatment is eventually carried out. With the patient in the upright position the changes seen in the spine will depend upon the degree of pain which the individual is having, especially back pain with associated muscle spasm. It is not uncommon in the acute state to see these patients hold themselves in a rather severe side-bending position with associated flexion and flattening of the lumbar spine. The gait becomes protective as jarring has a tendency to increase pain, and sudden turning or twisting will certainly aggravate it. It is well to examine the patient not only in the upright position but also in the prone position to determine the degree of fixation which may be present in the lumbar spine. Psoas spasm will produce a flattening and rigidity of the lumbar spine which will not subside with the patient prone. Many times these patients will not tolerate this position and will ask if they cannot lie on the side or back during this phase of the examination. There may be an obvious inequality in the two sides of the pelvis with an associated change in the shoulders as well. If these patients are asked to put themselves through a range of motion which might be considered normal, their motions are usually found to be very slow and deliberate, and any motion which tends to stretch the sciatic nerve is markedly limited.

It is necessary to have the patient locate the exact center of the pain as far as he is able in order to determine and trace the course from the origin to its most remote distribution. Many times the pain will start in the region of the lumbosacral articulation, and at other times the patient will tell you that the pain really starts in the hip and only traverses the posterior thigh to the knee. This distribution as well as the origin and extent of pain is certainly a variable one, and any part or all of the sciatic nerve may be affected.

Usually patients will also complain of a distinct tenderness over the spinous processes of the fourth and fifth lumbar vertebrae although this is not always true, and there may be no soreness anywhere in the lower lumbar spine. The descriptions are varied, depending upon the individual patient's ability to describe his symptoms. Most patients will say their backs hurt. Since the pain of this condition is one of a distributed nature over the course of the sciatic nerve, variations are found, and it is well to question the patient thoroughly as to the extent, duration, and type of pain present. It is also advisable to question the patient on other sensory manifestations of posterior root injury. The description will certainly vary as the sensory manifestations vary. One patient may state that the pain is exceedingly severe but, aside from the pain, the outside of the calf may be numb; there may be a sensation of tingling or prickling in the whole lower extremity; the lower extremity may feel heavy and dead; there may be numbness in one toe; or the leg may feel cold or hot. It is certainly advisable to elicit these various evidences of sensory nerve root changes and record their locations. It is also advisable to ask the patient the effect of sneezing or coughing and to determine whether the response is one of acute muscle spasm in the low back or whether the response is one of an exaggeration of pain down the course of the sciatic nerve. It is also well to ask if there is difficulty with bowel movements and if straining at stool increases the pain down the lower extremity or back. The effects of position are also inquired about, such as exaggeration of pain by hyperextension of the spine or side bending, and what effects, if any, ordinary exertion has upon back and leg pain. Some patients will tell you that by lying down they can soon be free of pain, and others will tell you that lying in the supine position has a tendency to increase pain and that they get relief only by being up and walking around. This is a variable reaction but, nevertheless, one that should be noted in the history at the time of examination.

I cannot too strongly emphasize the importance of a detailed and complete physical examination in suspected disk lesions. It is this examination that will in most cases determine the clinical diagnosis and, to a certain extent, indicate the procedure to be followed in treatment. Although these individuals vary in their response, there are several rather reliable signs which should always be looked for. Lower lumbar muscle spasm is a rather constant sign in the presence of acute disk rupture and will necessarily limit lower lumbar motion. It is always well to determine the degree of flexion of the lower lumbar spine with the knees held in extension and, at that time, to determine the severity and increase of sciatic nerve pain and to note the limitation of forward flexion of the spine. It is also well to determine the degree of extension of the spine upon the pelvis and to note the rapidity with which an increase in sciatic nerve pain is brought out. Side bending both to the right and to the left should always be carried out; usually bending to the side of pain will exaggerate it, and bending away from the pain will lessen it to some extent. This is not a completely reliable test, however, as I have seen the exact opposite occur in many patients. Rotation of the lower lumbar spine does not usually influence to any great extent the degree of pain as the rotatory changes are so limited at this particular time.

Points of local tenderness are, of course, sought for and recorded, and usually with lower lumbar ruptures, an exquisitely tender point will be noted directly over the spinous process of the fourth or fifth lumbar vertebra. It should also be noted that pressure directly lateral to the spinous process over the site of pain will increase the pain down the extremity in a few seconds. It is to be remembered that these signs are variable and that diagnostic acumen is necessary if a conclusive diagnosis is to be reached. The straight leg-raising test is always utilized; it is one of the simplest and most reliable clinical methods of detecting pain of root origin. The patient lies completely supine with the knees in full extension while the examiner raises one leg by flexing the hip with the knee maintaining its position of extension. Normally it is possible to raise the leg to approximately 90 degrees in this manner without causing discomfort other than a feeling of popliteal or hamstring pull. However where a definite disk rupture is present at either the fourth or fifth lumbar vertebra with nerve root compression, the degree of straight leg raising will be materially diminished and any attempt to force the leg beyond the point of fixation will be met immediately with in-

creased and marked resistance, but the degree of limitation will depend upon the individual and the severity of the rupture.

Cram[4] described a sign which he termed the posterior tibial nerve sign. This test is a modification, or rather an accentuation, of the straight leg-raising test, and is performed with the patient in the supine position, the leg being raised in the straight manner, first on the affected side and then on the sound side. When the leg has been raised to a point where pain is produced, the knee is flexed about 20 degrees and the leg is then further raised to a point just short of that which again causes pain. At this time firm pressure is applied to the middle of the popliteal space over the posterior tibial nerve, and if the test is positive, a rather sharp pain will be felt in either the lower lumbar spine or the affected buttock. In the sound leg the test either causes no pain at all or causes lower back pain with pain referred to the affected hip. Lasègue's test is certainly reliable, but it has the disadvantage that two angles are involved, and, as a consequence, the findings are more difficult to record accurately. This test is performed with the patient supine, and the leg is raised by flexing both the hip and the knee. When the hip has been flexed to 90 degrees, the knee is then slowly extended. Normally the knee can be fully extended with the hip in this position, but in the presence of root irritation, extension of the knee is limited, and severe pain occurs at the extreme of this movement. A further elaboration of these tests is produced by raising the thecal and root tension by flexing the cervical spine at the same time the straight leg-raising test is being performed. This will usually produce acute and extreme pain where the root is being stretched over a retropulsed disk. Dorsiflexion of the ankle with the leg in the position of extension while being raised in a straight manner will also exaggerate root pain owing to an exaggeration of the stretching force applied to the sciatic nerve.

The lower extremity reflexes are always checked and notation made of any change from normal. Changes in the patellar reflexes are not too common although in the presence of space-occupying lesions at either the third or fourth lumbar vertebra some diminution may be noted. Changes in the ankle jerk or the Achilles reflex are much more common in lower lumbar disk ruptures, and with a rupture of the lumbosacral disk the Achilles reflex is usually diminished, if not completely obliterated. It must be remembered, however, that this is again a variable manifestation, and the reflex may be absent one day and present the next. However, once this reflex has been completely lost, it is not uncommon, in spite of early surgery, to have a number of months or a year or more go by before a return is noted; many times it is permanently lost. The degree of diminution or change, of course, is noted in comparison with that on the sound side and also in comparison with the upper extremity reflexes of the individual. It is usually well to determine the reflex responses of the individual by testing the upper extremity reflexes first.

Other evidences or sensory changes such as hypesthetic or paresthetic disturbances are noted and recorded, and, if possible, the dermatome pattern is examined in the hope that the level of disk rupture may be determined clinically. There is such a great degree of dermatome overlay or overlap that it is exceedingly difficult to designate the specific nerve root involved in these cases. It is not uncommon, of course, to see actual motor changes take place which involve the muscles of the extremity affected, in producing either frank atonic or atrophic changes.

The jugular compression test is another test which may help clinch a clinical diagnosis of an extradural compressing lesion, and although it is not positive in a great percentage of cases, the response is usually spectacular in those cases where a frank herniation of the nucleus pulposus into the spinal canal is present. The same is true in the presence of a caudal tumor, but in my experience this test has been positive in only about 15 to 20 per cent of those cases with true disk ruptures. The test is performed with the patient in the sitting position; the jugular veins are compressed either by the use of a blood pressure cuff or by manual compression. The response, if positive, will usually be manifested in 10 to 20 seconds, with an increase of existing pain or some other sensory manifestation over the course of the sciatic nerve involved. Occasional patients have an irritable carotid sinus and, as a consequence, the physician must watch for changes from direct pressure over the sinus and not be too alarmed if an acute seizure results.

The presence of sacroiliac or hip joint disease should be ruled out; generally this can be accomplished by x-ray examination. This is one of the most important phases of the examination and serves as one of the chief mechanisms whereby a complete and adequate diagnosis is eventually arrived at. Every patient should have the benefit of a complete x-ray of the lumbar spine and pelvis, not only in the anteroposterior and lateral positions, but in the oblique position as well. Facet disease and actual pedicle disease can at times be seen only in the oblique position, and certainly it is a mistake not to include this as a routine in all low-back studies where any space-occupying lesion is being considered.

The male and female pelvis should always be examined as a part of the routine diagnostic survey of the suspected disk lesion. While pelvic disease is one of the uncommon causes of true sciatic pain of root origin, occasionally there is that case that will act as a background for sciatic neuralgia, and of course this should not be overlooked. If x-ray examination has not revealed any frank significant spinal disease, such as spondylolisthesis, true rheumatoid disease of the spine, tumors of the vertebrae, Paget's disease, or osteomyelitic or other inflammatory disease of the vertebrae, which could account for nerve root pressure, and if all other indications are pointing to a space-occupying lesion as the cause of the sciatica, myelography should be performed in order to confirm the clinical diagnosis.

In my opinion myelography should not be considered a prime mechanism in arriving at a conclusive diagnosis of the space-occupying lesion. I feel that myelography should be definitely placed in a secondary position and that the diagnosis of disk lesions should be made on the clinical basis and myelography be reserved for confirmatory procedure only. Certainly in experienced hands myelography is not the painful ordeal Dandy[5] once considered it to be but is a procedure that can be done easily and expeditiously with no pain or discomfort to the patient. The valuable feature of myelography is that it can and does confirm the location of the lesion, thus saving the need for exploratory surgery at a later date. As was previously mentioned,

without myelographic help it is almost impossible to determine specifically by the dermatome involved the exact level of the lesion.

There are, of course, the broad principles which would indicate whether a lesion is located at the fourth or fifth lumbar vertebra; change in the Achilles reflex is indicative of a lesion at the fifth lumbar vertebra and some diminution of the patellar reflex is indicative of a lesion at the fourth lumbar vertebra. However, the location can most easily and readily be confirmed by myelography.

Keegan[6] has stated that myelography is not needed at all and that the dermatome pattern should be the chief index for determining the source of nerve root irritability. Perhaps for Keegan this is true, but I have found it more convenient and suitable to confirm the location by myelography. It has apparently become the practice of some doctors to substitute myelographic procedures for a good thorough clinical examination and an associated accurate and detailed history; I feel that this is entirely wrong. Myelography, of course, was designed to reveal extradural displacing lesions and was not meant to be considered as a diagnostic agent for all the ills to which man's low back apparently is falling heir. It will also reveal intradural tumors and other intradural or subarachnoid adhesive processes, but for the sake of the subject at hand myelography is considered as a confirmatory diagnostic procedure in the localization of disk lesions.

When myelography is used to confirm a clinical diagnosis of rupture, minor defects on the film indicating a disturbance in position of nerve roots become exceedingly important and must have the benefit of the combined judgment and knowledge of the radiologist and the physician carrying out the myelographic procedure for correct interpretation. Radiologists usually report their findings directly from the film without having the knowledge of the patient which the surgeon would have after a detailed examination. As a consequence, the radiologist would certainly not attach the same significance as the surgeon would to minor changes in the contour of the oil column. Viewed in this respect, myelography becomes an exceedingly valuable aid to the surgeon in confirming or disproving his clinical diagnosis.

Having arrived at a clinical diagnosis of a ruptured lower lumbar disk and having this diagnosis confirmed by myelography, it becomes necessary to determine the type of treatment. It might be said that all patients should have the benefit of conservative treatment prior to surgical exploration, but, unfortunately, this is sometimes neither desirable nor economically feasible. When a patient is manifesting evidence, however, of the typical sciatic nerve syndrome of possible disk origin, but is not suffering excruciating pain requiring narcotics for relief, and his ability to ambulate is not impaired, it is probably wise to institute conservative treatment, at least for a trial period. Most surgeons have seen cases of apparent ruptured lower lumbar disks with the typical sciatic distribution of pain which have responded to conservative management and likewise have seen cases of a similar nature fail to respond to conservative treatment. However, I feel that it is incumbent upon surgeons to give the patient the benefit of the doubt and treat him as we ourselves would like to be treated.

If surgery were the last word in the management of the disk syndrome and would relieve successfully every patient it should certainly be performed in all cases. Since there is no one particular type of treatment which will completely and satisfactorily handle all cases, it is only good judgment, if the patient can tolerate it, to try conservative treatment over a reasonable period.

By conservative treatment I mean office treatment of a manipulative nature with associated diathermy or some other form of deep heat, analgesics as needed, traction, hydrotherapy, and perhaps the use of intravenous calcium to add to the patient's comfort. Supportive measures such as wearing a lumbosacral corset during the day and having a proper support in the mattress during sleeping hours are usually indicated. In the acute state of the ruptured lower lumbar disk a certain degree of joint instability is also present, and successful treatment must include proper support during this stage. There will be a certain percentage of these patients who will respond to conservative office treatment and whose symptoms will clear up, some in an exceedingly short time and others over a longer period, that is, in several weeks. I think in most of these instances in which the patient is suffering a distinct sciatic neuralgia in which conservative treatment is apparently not successful, or at least if he is not showing definite improvement in 3 to 4 weeks, other measures should be instituted. If the patient is going to respond, usually some favorable response will start in 3 to 4 weeks so that both patient and physician will be aware that changes are taking place which are likely to lead to ultimate complete relief. It is as exceedingly difficult for me to believe that anyone would assume that all cases can be cured by conservative management, manipulative therapeutics, or any other type of nonsurgical treatment as it would be for me to assume that all cases receive 100 per cent benefit from surgical management.

I feel it wise to reserve for surgery those cases which fail to respond to conservative office treatment, and in a true disk rupture this percentage is rather large. Anyone who has seen many extradural extrusions of the nucleus into the spinal canal with adhesive changes to the nerve root certainly appreciates to the full extent the utter impossibility of receiving complete relief by manipulative therapy.

Those cases which apparently will show a favorable response by increasing the stability of the joint by whatever method the surgeon might deem advisable are those in which the posterior longitudinal ligament is intact and only a slight retropulsion is present into the spinal canal. Many cases have been surgically fused without actually removing a disk, and the patient has received a marked degree of relief. Relief has also been obtained without fusion and without actually removing the disk by merely decompressing the nerve root. However, I believe that today most surgeons doing disk surgery are removing the disk as completely as possible as well as decompressing the nerve root or doing a partial foraminotomy and completely removing any extradural pressure which might be exerted on the root from a hypertrophied subflavum ligament.

This is the technic I have followed for the last several years, and it has been eminently successful insofar as any one particular method could be for this condition. Only a small portion of the adjacent laminae is removed at surgery, but I make it a practice to remove as completely as possible the subflavum ligament and to make absolutely sure that the nerve root itself

has been completely decompressed. This, of course, requires that the nuclear substance of the disk be removed as completely as possible, and by this I mean that following the removal with pituitary rongeurs, the cartilaginous plates are completely cleaned and the bony plates slightly eroded. I believe it is anatomically impossible to remove all of the disk at surgery, and since the nucleus is the great troublemaker in these cases, it is the structure I attempt to remove cleanly. If fibrous fusion takes place between the bodies of the vertebrae, and I feel that it does, I believe it is particularly important that the cartilaginous plates, which act as part of the confining structures of the nucleus, should be completely curetted.

I incise the posterior longitudinal ligament longitudinally, that is, parallel to the fibers of the ligament rather than in the transverse manner. I feel that when healing takes place, if there should be part of the nucleus left, the tendency to extrusion would be minimized by making the healing process easier by a longitudinal incision. In many cases there is already erosion through the ligament and extrusion of nuclear material, but the opening usually has to be enlarged somewhat, and I do not believe the technic which removes completely the portion of the ligament overlying the annulus is good surgical procedure.

Without going into extensive detail regarding the technic of surgical treatment, suffice it to say that the method I have briefly described is one that seems to work best and produce the most satisfactory results for me. I might say that when meticulous care is used in approaching the facets laterally, the capsule is not stripped of its fibers and the facet itself is not surgically interfered with. If surgical fusion is not planned following disk resection, great care should be taken to make sure that no damage has been produced in the facet structure itself.

SUMMARY AND CONCLUSIONS

The chief indication for surgical management of the ruptured lower lumbar disk is failure to respond to conservative management. Failure of conservative treatment is difficult to assess just as failure of surgical treatment is sometimes exceedingly difficult to assess, to analyze properly, and explain. The temperament of the individual, his occupation, and his habits certainly must be considered.

The one circumstance in which the surgical indications are very definitely clear is the massive extrusion of the nucleus into the spinal canal with pronounced cauda equina pressure. This, of course, is not a common emergency but one that will be met with occasionally by surgeons who see much of this type of work. Certainly with typical cord, bladder, and bowel involvement produced by this mechanical defect, time should not be wasted with conservative measures.

When a nerve cell dies it will not regenerate, and pressure on a nerve root over a long period undoubtedly produces death of some nerve cells, interferes with the conduction mechanism of the nerve, and produces irreversible changes in distribution in the extremity. Atrophic changes are seen which become permanent, especially in the muscles of the buttock and the thigh, and even after the pain has been relieved by surgery some dysfunction resulting from the previous pressure remains. Because of this loss of permanent muscle tone and atrophic change, changes in gait and secondary postural changes in the lower back which add to joint and muscle strain are not uncommon.

When to operate and when not to operate become important factors in the evaluation of these patients, and how long to try conservative treatment is certainly one of the problems that confronts the physician when he accepts a case for this type of therapy. I feel that he can definitely tell in most patients within 2 to 4 weeks in which direction the condition is going, that is, whether the patient is making a distinct degree of progress, the condition is remaining stationary, or actual harm is occurring as a result of delay. Muscle weakness and wasting in particular indicate rather severe interference with root function, and if compression is allowed to persist too long, irreversible changes almost invariably will occur.

Contraindications to operation in suspected ruptured lower lumbar disk must also be considered. Confused, inadequate, or doubtful diagnosis should be a prime consideration of the surgeon, and if there is a question of doubt, it is probably better in most instances not to consider surgery but to institute extensive conservative management. The neurotic patient is a second contraindication. This again becomes a question of the individual surgeon's experience in evaluating and placing proper emphasis on true pain of nerve root origin and so-called pain expressed by a patient in a frank state of neurosis. Every surgeon has probably seen these patients and has made the mistake at some time of operating on a patient who would have been much better off under continued conservative or even psychiatric care. The neurotic patient perhaps is best treated by the conservative method.

In conclusion I would state that a detailed history, meticulous physical examination, and conclusive diagnosis are the prime prerequisites to treatment of a ruptured disk. In the average patient whose condition is not too severe and in whom the diagnosis has been established, I feel that a reasonable trial of conservative management should be made. If normal progress is not apparent in 3 to 4 weeks, I would then not hesitate to advise surgical exploration. With this type of management I have had exceptionally gratifying results. The negative surgical explorations are indeed minimal, and the great majority of patients receive marked relief from their pain with a relatively short convalescence after surgery. The average patient returns to his usual occupation within 6 to 7 weeks after discharge from the hospital when meticulous nontraumatic surgery follows a correct diagnosis of a ruptured lower lumbar disk.

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Current Medical Literature

THE USE OF IPRONIAZID IN THE TREATMENT OF BONE AND JOINT TUBERCULOSIS

The experimental use of Iproniazid on 66 patients with tuberculosis of bones and joints is reported by David M. Bosworth, M.D., and his coworkers in *The Journal of Bone and Joint Surgery*, July, 1953.

The ages of the patients in this study ranged from 6 months to 80 years, although one third of the group was between 40 and 50 years. The duration of the experiment was slightly more than 1 year. Five patients died during treatment; however, there was no evidence of Iproniazid toxicity in any case. Streptomycin had been administered to 53 patients, aureomycin, terramycin, Isoniazid and streptokinase had also been given in some cases. All other medication was stopped when Iproniazid therapy was begun.

All but 9 of the 66 patients gained weight; none lost weight except as a terminal manifestation. The average weight gain was 15.3 pounds.

There were 56 sinuses among 38 patients, 35 of which healed and remained healed. Twenty-one improved to a stage of intermittent healing, dry crusting, or reduced drainage. Five presinus lesions subsided and healed.

Of 35 patients with open wounds, all but 4 showed healing or decided improvement. Iproniazid seemed to bring about improvement in closed abscesses. A change in the bloody content of the discharge took place in some lesions. In 27 patients with open lesions negative cultures were obtained throughout the treatment period, or their incisions healed and no further cultures could be obtained. In 3 patients there was one positive culture among many negative cultures. In 1 there was an occasional positive culture interspersed with many negative cultures. Other benefits obtained with Iproniazid were a satisfactory temperature record and a relief of pain in the majority of patients.

Sinuses reacted to Iproniazid with a thinning or reduction of discharge, absence of granulations or reduction to below the surface of the skin, and reduction of local edema and local heat.

Numerous exhibitions of toxicity are evidenced by patients undergoing Iproniazid therapy. With the exception of psychosis, all are mild or correctable. Psychoses are uncommon, appear to be reversible, and can probably be avoided by reducing dosage. Other evidence of toxicity included dizziness, tremor, presence of a Babinski reflex, hypertonic reflexes, clonus, euphoria, drowsiness, headaches, tinnitus, coma, mouth dryness, diaphoresis, weakness of visual accommodation, hesitancy, obstipation, impotence, flushing, twitching, and paresthesia.

Laboratory findings in most cases were normal. Only 9 of 52 patients having liver function tests performed showed any signs of liver damage.

No complications or adverse reactions were noted in any of the 23 patients receiving anesthetics or preoperative medication. No operative or postoperative complications occurred. This tends to refute reports that anesthesia, preoperative medication, and operative procedures might prove dangerous for patients receiving Iproniazid.

Twenty-nine of the 66 patients in the study are healed and medication has been discontinued. Twenty-five are still on medication; 5 of these are healed; 20 are improved. Three who are improved were taken off medication. Five patients died and another probably will. In 2, treatment lasted less than a week. One patient has shown no signs of improvement. Two children with proved lesions of the knee have recovered and are fully ambulatory with essentially normal knees; the drug has been discontinued. Although it is too soon to be certain, it seems probable that the lesions are healed and the joints mobile.

Complete knowledge of the benefits and toxicities arising from the use of Iproniazid is still not available and further investigations must yet be made before a complete evaluation can be obtained.

COMMON WHIPLASH INJURIES OF THE NECK

A study of 50 persons who sustained whiplash injury of the neck as a result of a rear collision between automobiles has been reported by James R. Gay, M.D., and Kenneth H. Abbott, M.D., in the *Journal of the American Medical Association*, August 29, 1953. None of the group received a direct blow on the head or neck.

A rear-end collision of automobiles results in sudden and forceful flexion of the neck which is sometimes followed by less forceful snapping of the neck in alternate flexion and extension. In the series reported the injury resulted in mechanical trauma of the supporting ligaments of the cervical spine, and seriousness of the injury depended on the number of complications and their variety.

Cervical radiculitis was the most frequent complication, occurring in 35 patients. In 31 patients there was evidence of cerebral concussion, and in them nervous symptoms were particularly intense. Concussion probably occurred because of sudden mechanical deformity and pressure on the frontal and temporal lobes of the brain as a result of arrest of the forward movement of the brain against the anterior walls of the skull as the head and neck were snapped back after acute flexion. Lesser concussions probably also occurred in the frontal, temporal, and occipital lobes as a result of alternate flexion and extension. The effect of acceleration and deceleration on brain tissue may be another important factor.

Protruding intervertebral disk in the cervical region occurred in 13 patients. Low-back injury was present in 15.

Roentgenograms showed hypertrophic arthritis of the cervical spine in five patients, subluxation of the lower cervical spine in one patient, and a laminar fracture in another.

A persistent psychoneurosis was present in 26 patients, and the authors believe that the existing circumstances of a whiplash injury tend to development of an emotional reaction. Psychoneurotic reaction was absent or mild if patients were examined, told of their injuries and what to expect of them, and placed on a definite treatment program within a few hours or days of the accident.

Therapy included an explanation to the patient of the nature and variety of injury. Emergency hospitalization was recommended to those with acute injuries when the patient was disturbed emotionally, suffered a moderate concussion, or was known to have subluxation, fracture, or dislocation of the cervical spine.

The patient was managed at home in cases of acute, mild, or moderate whiplash injury, and rest in bed for 24 to 72 hours was prescribed.

Intermittent cervical traction was instituted in cases of severe muscular spasm and radiculitis. During the acute phase the patient was in traction for 2 hours and out of traction for 2 hours; during convalescence 1 hour of traction several times a day was prescribed.

Warm baths were used to combat tension symptoms, and daily activities were controlled. Attempts were made to distract patients from preoccupation with their injuries by encouraging manual activities.

A psychoneurosis alone, in some cases probably contributed to by the legal aspects of the case, or a psychoneurosis in combination with a somatic condition was the cause of delay in improvement in the greatest percentage of patients.

WHAT THE CLINICIAN SHOULD KNOW ABOUT HYPOXIA

Homicidal apathy toward what should as a matter of course be familiar to all practitioners—etiology, diagnosis, and correct emergency treatment of hypoxia—the reasons for it, and suggestions for overcoming it are presented by Paluel J. Flagg, M.D., in the September, 1953, *American Practitioner.*

Assigning asphyxiated persons to the care of rescue squads, thus implying that circulatory stimulation is the only thing that can be done; failure to realize that disconnected accidents involving hypoxia in reality have a common bond of treatment; and confining recent developments in cardiorespiratory physiology to experimental departments may be the reasons for this apathy.

It is suggested that instruction be provided the undergraduate student and the general practitioner; the thoracic surgeon, the anesthesiologist, and the general practitioner-anesthetist are most qualified to provide this.

The symptoms of asphyxia should be familiar so that an instant diagnosis of the degree can be made and immediate action instituted. Each degree has its own indications for treatment and it should be remembered that the asphyxial state is not static; it may change quickly from one degree to another. Ability to evaluate and classify resuscitation apparatus through intended effects is important.

In an attempt to make instruction in the technic of instrumentation easily available to the general practitioner in a short period, intensive week-end courses have been given in New York since 1947 and in other cities as well. Courses are given under the auspices of the Society for the Prevention of Asphyxial Death, Inc. The groups in New York, which are available to out-of-town students, meet the first Friday and Saturday of each month at the New York Academy of Sciences, 2 East 63rd Street. Details concerning the courses may be obtained from the secretary of the Society.

THE CLINICAL SIGNIFICANCE OF THE INCREASING RESISTANCE OF ORGANISMS TO THE ANTIBIOTICS

The rapidity with which more and more organisms are becoming increasingly resistant to antibiotics through indiscriminate use and the simple and rapid means available for determining bacterial sensitivity to the various antibiotics are described by Frank L. Meleney, M.D., and Balbina A. Johnson, B.A., in the September, 1953, issue of *Surgery, Gynecology and Obstetrics.*

Various organisms have built up resistance to the sulfonamides and antibiotics as each has appeared in the medical armamentarium. Whereas sulfonamides were effective in hemolytic streptococcal infections, they were ineffective in staphylococcal infections. Strains of bacteria developed which were resistant to sulfanilamide, to penicillin, or to streptomycin. Penicillin, which was originally thought to be a panacea, soon, with the development of resistant strains of bacteria, required greater dosages to combat infection. Indiscriminate use of, self-treatment with, and inadequate dosages of penicillin caused the organisms to be held in check instead of being destroyed. The bacteria which were resistant to the antibiotic in turn produced bacteria which were even more resistant. In addition, certain resistant organisms produced penicillinase which is capable of destroying penicillin, thus interfering with its bactericidal and bacteriostatic action. One authority has gone so far as to state that this increased resistance has been demonstrated with every antibiotic and bacterial species studied. There is evidence that the bacterial resistance is built up in proportion to the frequency of and indiscriminate use of the antibiotic. With the broad spectrum antibiotics, a cross-resistance is built up—a resistance to one builds up a resistance to the other, particularly in the case of aureomycin and terramycin.

The authors performed laboratory tests to determine the sensitivity of various bacteria to penicillin, bacitracin, streptomycin, terramycin, and neomycin. The organisms obtained, in almost every species, were more resistant from inpatients than from outpatients. A number of methods have been devised for testing sensitivity. Described are the medicated disk, tube dilution, turbidimetric, and plate dilution methods. These tests, by indicating which drug the organism is susceptible to, indicate which antibiotic or combination of antibiotics is the most potent and should be employed. The medicated disk method has been selected by most investigators and laboratory workers because it is simple, rapid, and consistent. It is hoped that with the use of these tests antibiotics will be more discriminately selected and administered.

A PRACTICAL DEVICE FOR LEG TRACTION AT HOME

A traction device for home use that can be made for approximately $8.00 is described by Angelo M. May, M.D., and G. F. Norman, M.D., in *California Medicine,* June, 1953. Hospitalization in itself is so expensive that it seems impractical for leg traction alone, and pulley traction apparatus set up in the home has proved cumbersome, difficult to manage, and tends to destroy furniture. The device which is described uses the lever principle rather than the pulley.

The lever-type traction device is in the form of an isosceles triangle constructed from a 1 by 4-inch board frame put together with ordinary hinges. A heavier hinge is used at the angle farthest from the bed in order to supply the weight for traction. A regular 3½-pound gate hinge will usually supply the necessary amount of pull. A hinge with a removable pin attaches the traction frame to an ordinary bedboard placed under the mattress. The bed can easily be made when the frame is not in use by withdrawing the pin from the hinge and detaching the device from the bedboard. Adjustment of the weight of pull is accomplished by changing the degree of the obtuse angle formed by the bedboard and the side of the device hinged to it. A spring scale may be placed in the line of traction if it seems desirable to measure the amount of traction.

The leg is prepared for traction by placing sponge rubber pads along its medial and lateral surfaces and placing a strip of web belting with a flannel covering over the sponge rubber, with the ends of the strip on either side of the leg and a loop below the foot. The rubber and belting are held in place with an Ace bandage, and the loop of the sling is attached to the board frame. A "loafer" type shoe tends to prevent the web belting from pressing upon the ankle.

SEDATION WITHOUT DEPRESSION IN THE MANAGEMENT AND TREATMENT OF THE AGED

Statistics are not necessary to point out to the physician the tremendous increase in population of persons over 65, but articles such as that by Henry V. Grahn, M.D., in the *American Practitioner,* August, 1953, advocating more than advice and heavy sedation for aged patients, are of value to all.

Many of the problems of geriatrics are specific and their solutions are handled by the specialist. However, many problems of the aged which apparently have no organic basis—weariness, languor, feelings of rejection and isolation, anxieties over things large and small, and depressions—are dismissed as due to old age or treated with barbiturates and sympathetic advice.

The value of sympathetic understanding and advice for old people is not to be underestimated, but for them barbiturates act as depressants rather than sedatives. In order to obtain daytime sedation without drowsiness, agitation, or depression, the author administers ½ grain amobarbital and 5 mg. d-amphetamine sulfate in combination (Dexamyl) to elderly patients whose complaint is emotional inflexibility. Administering these two drugs together counteracts the individual stimulative or sedative effect and enhances the antidepressant effect of each.

Three case histories typical of thirty-five patients treated with amobarbital and d-amphetamine are presented. Careless diagnosis is not recommended, need for sympathetic advice is pointed out, and improvement of mood and production of tranquility without drowsiness and depression are stressed.

BOOK NOTICES

(Continued from page 236)

four divisions—Principles of Nutrition, Diet in Disease, Food Selection and Preparation, and Tabular Material and Special Tests—each chapter within the division gives the basic sources of the subject matter and completely covers its usage. An unusually fine list of selected references including books, journals, and pamphlets has been included for further reading.

SYNOPSIS OF OBSTETRICS. By Jennings C. Litzenberg, B.Sc., M.D., F.A.C.S., Late Professor Emeritus of Obstetrics and Gynecology, University of Minnesota Medical School, Minneapolis. Ed. 4, revised by Chas. E. McLennan, M.D., Professor of Obstetrics and Gynecology, Stanford University School of Medicine, San Francisco. Cloth. Pp. 378, with illustrations. Price $5.50. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1952.

Designed as a quick reference for essential material in obstetrics this Synopsis follows closely the presentations in standard textbooks covering the specialty. This fourth edition, revised by a former associate of Dr. Litzenberg, adheres to the outline of the first three manuals. The chapter headings are divisions of the following large topics: anatomy, physiology and development of the ovum, physiology of pregnancy, and conduct of pregnancy. In order to incorporate the advancements made in obstetric care during the last 5 years, some deletions were made and certain major sections, particularly those concerned with the early life of the ovum, endocrine physiology, RH isoimmunization, regional anesthesia, roentgen pelvimetry, and pregnancy toxemia, were completely rewritten. It is important in a book of this type to have a good visual presentation; therefore, new illustrations have been added; all of the 157 illustrations are adequately labelled. As a book to consult this small, concise, and well-indexed manual should be on the desk of all physicians.

OPERATING ROOM TECHNIC. St. Mary's Hospital, Rochester, Minnesota. Ed. 4. Cloth. Pp. 345, with illustrations. Price $6.50. W. B. Saunders Company, West Washington Square, Philadelphia, 1952.

Recent advances in operative surgery have made it necessary to rewrite completely this fourth edition of "The Operating Room—Instructions for Nurses and Assistants" which was first published in 1924 and revised for the third time in 1937.

The present edition is all a manual should be: Procedures for surgical operation are classified under general, orthopedic, and neurologic, and procedures in urology and peroral endoscopy are presented step by step. Inventories for sterile trays are included under special therapeutic procedures; and specific duties are listed for each member of the surgical team. The Mayo Clinic contributed the excellent photographs which are used throughout the book and which are supplemented by clearly labelled illustrations and diagrams. A detailed table of contents, a full index, and a special index of illustrated instruments are further aids in making this manual extremely useful to members of surgical teams.

A VITAMIN DIGEST. By Guy W. Clark, Technical Director, Lederle Laboratories Division, American Cyanamid Company, Pearl River, New York. Cloth. Pp. 254, with charts and figures. Price $6.50. Charles C Thomas, Publisher, 301-327 East Lawrence Ave., Springfield, Ill., 1952.

Requests for reprintings of a Lederle Laboratories Bulletin issued in 1942 which presented a condensed account of the various vitamins provided the stimulus for "The Vitamin Digest" by the technical director of the Laboratories. Mr. Clark devotes one chapter of the book to each of seventeen vitamins; the detailed description of each vitamin conforms to an established outline beginning with a discussion of the discovery of the vitamin, continuing with a description of its characteristics, physiologic effects and food sources, and ends with an extensive list of references. These individual chapters are well written and contain pertinent material gleaned from 40 years of research. Only two and one half pages are devoted to a summary in which Mr. Clark points out that we, the American people, have not attained the ideal nutritional goal because of refinement of foods and modifications of our diets. Not discussed are multivitamin deficiencies in which symptoms are more diffuse, more widespread, and more difficult to identify than those caused by gross deficiencies of specific vitamins. As an up-to-date reference book on vitamins this is recommended.

THE USE OF DRUGS. A Textbook of Pharmacology and Therapeutics for Nurses. By Walter Modell, M.D., Assistant Professor, Cornell University Medical College, and Doris J. Place, R.N., Instructor in Medical Nursing, Cornell University-New York Hospital School of Nursing. Cloth. Pp. 468. Price $4.50. Springer Publishing Co., Inc., 1 Madison Avenue, New York 10, N. Y., 1953.

When a doctor and a nurse collaborate in writing a book on a subject which is pertinent to each the result is gratifying. A new approach to the subject of drugs and their use correlates the practice of medicine and teaching, both authors being teachers as well as practitioners. This well-organized book makes use of all accepted teaching methods in presenting the subject of drugs and their use. The authors' approach, namely that adequate drug administration implies understanding of drug actions—both therapeutic and toxic, is emphasized again and again and is clarified both in the text and in diagrams. A valuable feature of the book is its division into four parts: Principles of Pharmacy, Principles of Therapeutics, The Medicine, Materia Medica (alphabetically arranged and classified for maximum usefulness). Each part is divided into chapters which in turn are divided by numerous subheads, simplifying the learning process as well as being conducive to easy reference. Particular pharmacologic or therapeutic problems are fully discussed in a single place. Since every phase of drug therapy is clearly outlined this book is valuable to all medical personnel as a quick reference to individual drugs or groups of drugs as well as for review of the fundamentals of drug therapy.

DIABETIC MANUAL for the Doctor and Patient. By Elliott P. Joslin, M.D., Sc.D., Clinical Professor of Medicine, Emeritus, Harvard Medical School; Medical Director, George F. Baker Clinic at New England Deaconess Hospital; Consulting Physician, Boston City Hospital; Honorary President, International Diabetes Federation; Honorary President, American Diabetes Association. Ed. 9. Cloth. Pp. 315, with illustrations. Price $3.00. Lea & Febiger, Washington Square, Philadelphia 6, 1953.

Dr. Joslin has again revised his well-known "Diabetic Manual for Doctor and Patient," now in its ninth edition, to incorporate recent advances in his specialty. For the doctor the manual can be used as a refresher or as a reference for specific problems in preventing, treating, and controlling diabetes. Five new chapters, on tuberculosis, neuritis, and complications of the eye (all in relation to diabetes); on the employment of and insurance for diabetics; and on the objectives and facilities of the American Diabetes Association, have been added. Osteopathic physicians will approve of the author's emphasis on the maintenance of a normal physiologic state which is the principle that underlies all else in the book.

The manual, in simple, easy-to-understand language, thoroughly guides, instructs, and informs the patient in the management of his disease. Because Dr. Joslin believes that an informed patient is apt to be a cooperative one, he has used various technics in teaching: prose, questions and answers, diagrams and charts, and tables, thus repeating in various forms all essential knowledge.

The book then is all the title implies—a diabetic manual for the doctor and patient; such a serious disease as diabetes requires the cooperation of both. It should be recommended to *all* diabetic patients to read and consult.

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Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Eighth Annual Convention, Royal York and King Edward Hotels, Toronto, Canada, July 12-16, inclusive. Program Chairman, Campbell A. Ward, 51 Cass Ave., Mt. Clemens, Mich.

American College of Osteopathic Internists, annual meeting, Philadelphia, October 7-10. Secretary, Glennard E. Lahrson, 460 Staten Ave., Oakland 10, Calif.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Hotel Statler, Los Angeles, February 16-19. Secretary, Arthur A. Speir, Box 57, Merrill, Mich. Program chairman, Richard E. Eby, 1247 N. Park Ave., Pomona, Calif.

American College of Osteopathic Surgeons, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Administrative Secretary, Mrs. O. F. Martin, Box 474, Coral Gables 34, Fla.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8. Program Chairman, Walter R. Garard, 809 S. Hobart Blvd., Los Angeles 5.

American Osteopathic College of Anesthesiologists, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, Crawford M. Esterline, Box 155, Kirksville, Mo. Program Chairman, Armin L. Karbach, 316 N. Center, Arlington, Tex.

American Osteopathic College of Radiology, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, H. Miles Snyder, Art Centre Hospital, 5435 Woodward Ave., Detroit 2. Program Chairman, John H. Pulker, Flint Osteopathic Hospital, 416 W. Fourth Ave., Flint 4, Mich.

American Osteopathic Hospital Association, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Executive Secretary, Mr. Robert P. Chapman, 1011 Kahl Bldg., 326 Third St., Davenport, Iowa.

American Osteopathic Society of Proctology, annual meeting, Hotel Jefferson, St. Louis, Mo., April 29-May 1. Secretary, Carl S. Stillman, Jr., 3523 Fifth Ave., San Diego 3, Calif. Program Chairman, Felix D. Swope, 609 Farragut Medical Bldg., Washington 6, D. C.

Colorado: See Rocky Mountain Osteopathic Conference.

Florida, annual meeting, Miami Beach, May 13-15. Secretary, Dominic Raffa, 5009 Central Ave., Tampa 3. Program Chairman, William E. White, Manatee Post Office Arcade, Bradenton.

Georgia, annual meeting, Radium Springs, May 7-8. Secretary, Kenneth H. Wiley, 806 Mortgage Guarantee Bldg., Atlanta 3. Program Chairman, Chan L. Plair, 901 Second Ave., Albany.

Illinois, annual meeting, Pere Marquette Hotel, Peoria, May 2-4. Executive Secretary, Mr. Douglas Durkin, 5200 S. Ellis Ave., Chicago 15.

Indiana, annual meeting, South Shore Inn, Lake Wawasee, Syracuse, May 9-11. Secretary, F. A. Turfler, Jr., 107 S. William St., South Bend.

Maine, annual meeting, Hotel Samoset, Rockland, June 17-19. Executive Secretary, Roswell P. Bates, 72 Main St., Orono.

New Mexico, annual meeting, Franciscan Hotel, Albuquerque, April 29-May 1. Secretary, Robert E. Smith, 205 N. First, Lovington.

Ohio, annual meeting, Columbus, May 2-5. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus. Program Chairman (annual meeting), Donald J. Ulrich, 141 E. Main St., Kent.

Oklahoma, annual meeting, Biltmore Hotel, Oklahoma City, November 8-10. Executive Secretary, Mr. Walter L. Gray, 210-12 Braniff Bldg., Oklahoma City.

Virginia, annual meeting, Williamsburg, May 28-29. Secretary, E. Brooks Flickinger, 301 W. Whitlock St., Winchester. Program Chairman, Vincent H. Ober, 407-11 Bankers Trust Bldg., Norfolk 10.

West Virginia, annual meeting, Daniel Boone Hotel, Charleston, June 6-8. Secretary, Guy E. Morris, 542 Empire

Bank Bldg., Clarksburg. Program Chairman, Donald C. Newell, 137½ Main St., Oak Hill.

Western States Osteopathic Society of Proctology, annual meeting, Shamrock Hotel, Houston, Tex., October 4-6. Secretary, E. F. Waters, 925 E. South Temple St., Salt Lake City 2.

Wisconsin, annual meeting, Schroeder Hotel, Milwaukee, April 12-14. Secretary, Edwin J. Elton, 1518 N. Seventieth St., Wauwatosa 13.

State and National Boards

ARIZONA

Those interested in professional examinations, contact Russell Peterson, D.O., secretary, Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

COLORADO

Esther B. Starks, Denver, has been re-elected secretary of the Basic Science Board. Her appointment to the Board will expire June 30, 1957.

CONNECTICUT

Professional examinations March 9. Address Frank Poglitsch, D.O., secretary, Osteopathic Examining Board, 300 Main St., New Britain.

Basic science examinations February 13, Room 23, Lampson Hall, Yale University, New Haven. Applications must be received no later than 2 weeks before examination. Address Miss M. G. Reynolds, executive assistant, State Board of Healing Arts, 52 Whitney Ave., New Haven 10.

DELAWARE

Examinations at Dover January 12-14. Applications must be filed on or before December 15. Address Joseph S. McDaniel, M.D., secretary, Board of Medical Examiners, 229 S. State St., Dover.

HAWAII

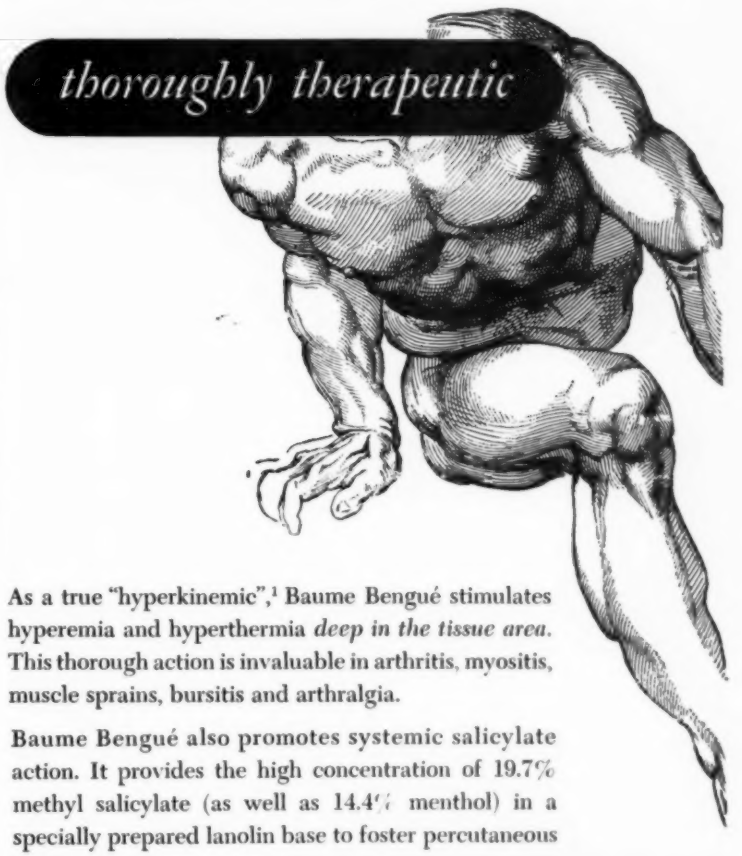
Examination dates by Territorial Law are usually the first Wednesday, Thursday, and Friday of January, April, July, and October. One of these dates is set for examination after approval of candidate's application by the Board. Address Frank O. Gladding, D.O., secretary, Board of Osteopathic Examiners, 504 Kauikeolani Bldg., Hawaii 13.

ILLINOIS

Examinations in January. Address Vera M. Binks, director, Department of Registration and Education, Springfield.

IOWA

Basic science examinations January 12, Capitol Bldg., Des Moines. Applications must be filed prior to examination. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.



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1. Lange, K., and Weiner, D.: J. Invest. Dermat. 12:263 (May) 1949.

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KANSAS

Examinations February 18-20, National Bank of Topeka Bldg., Topeka. Applications must be filed 30 days prior to the meeting. Address Forrest H. Kendall, D.O., secretary, Board of Osteopathic Examination and Registration, 420½ Pennsylvania Ave., Holton.

MASSACHUSETTS

Examinations January 12. Address Robert C. Cochrane, M.D., secretary, Board of Registration in Medicine, Room 33, State House, Boston 33.

MICHIGAN

Basic science examinations February 12-13, Detroit and Ann Arbor. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 410 W. Allegan, Lansing.

MINNESOTA

Carl E. Morrison, D.O., St. Cloud, has been reappointed to the Basic Science Board for a term expiring April 1, 1959.

MONTANA

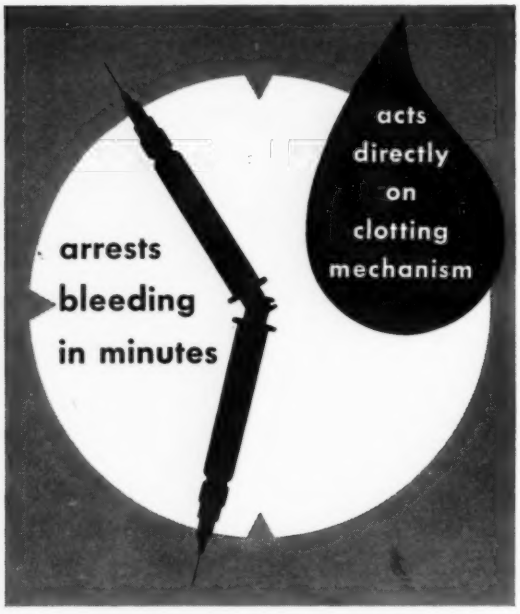
Examinations March 2. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEBRASKA

Basic science examinations January 12-13. Address Mr. Husted K. Watson, director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

NEVADA

Professional examinations January 30, 210 West Second Street, Reno. Applica-



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tions must be filed not less than 2 weeks before examination. Address Walter J. Walker, D.O., secretary, Board of Osteopathic Examiners, 210 West Second Street, Reno.

Basic science examinations January 5. Address Dr. Frank Richardson, secretary, Board of Examiners in the Basic Sciences, University of Nevada, Reno.

NEW HAMPSHIRE

Examinations March 10-11. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW MEXICO

Basic science examinations January 17. Address Mrs. Marguerite Cantrell, Board of Examiners in the Basic Sciences, P.O. Box 1522, Santa Fe.

NEW YORK

Examinations February 16-19. Address Stiles D. Ezell, M.D., secretary, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

NORTH DAKOTA

Examinations January 9-10 at Minot. Applications must be filed by December 15. Address G. L. Hamilton, D.O., secretary, Board of Osteopathic Examiners, 119 Main St. South, Minot.

OHIO

Examinations December 17-19. Address H. M. Platter, M.D., Wyandotte Bldg., Columbus 15.

OREGON

Examinations January 4-5 at the University of Oregon Medical School, Portland. Reciprocity and endorsement applications will be heard January 15. Address Mr. Howard I. Bobbitt, executive secretary, Board of Medical Examiners, 609 Failing Bldg., Portland 4.

RHODE ISLAND

Professional examinations January 7-8 at the State Office Bldg., Providence. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations February 10. Applications must be filed 20 days prior to the examination. Address Mr. Casey.

SOUTH DAKOTA

Professional examinations, Sioux Falls, 9 a.m., January 19. Address John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, First National Bank Bldg., Sioux Falls.

Beginning January 1, 1954, all South Dakota licenses to practice must be registered with the secretary of the Board of Medical and Osteopathic Examiners and renewed each year thereafter. Soon after January 1 notices will be mailed to all holders of South Dakota licenses. Holders of such licenses not receiving notices should contact the executive secretary. Address John C. Foster, First National Bank Bldg., Sioux Falls.

TENNESSEE

Examinations February 10 at Hotel Hermitage, Nashville. Applications must be filed by January 30. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

VERMONT

Examinations January 20-21 at the State House, Montpelier. Applications must be filed by January 10. Address C. D. Beale, D.O., secretary, Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

WASHINGTON

Professional examinations January 11-13 at the auditorium of the Health Science Bldg., University of Washington, Seattle. Applications must be filed 15 days prior to the examination. Address Robert L. Smith, director, Professional Division, State Department of Licenses, Olympia.

Basic science examinations January 6-7 at the auditorium of the Health Science Bldg., University of Washington, Seattle. Applications must be filed 15 days prior to the examination. Address Robert L. Smith, director, Professional Division, State Department of Licenses, Olympia.

WISCONSIN

Examinations January 12-14 at Madison. Applications must be filed by December 29. Address A. G. Koehler, M.D., secretary, Board of Medical Examiners, 46 Washington Blvd., Oshkosh.

WYOMING

Examinations February 1 at Cheyenne. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

REREGISTRATION OF OSTEOPATHIC LICENSES

December — District of Columbia, $2.00. Address Daniel L. Seckinger, M.D., secretary-treasurer, Board of Examiners in Medicine and Osteopathy, Commission on Licensure, Room 4130, Municipal Building, Washington, D.C.

December 31—Tennessee, $5.00. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland Ave., Jackson.

January—Alberta. No registration. Pay $10.00 a year membership in College of Physicians and Surgeons, Alberta.

During January — Connecticut, $2.00. Address Frank Poglitsch, D.O., secretary, Osteopathic Examining Board, 300 Main St., New Britain.

During January — Minnesota, $2.00. Address Wallace F. Kreighbaum, D.O., secretary, State Board of Osteopathic Examiners, 2748 Hennepin Ave., Minneapolis 8.

During January — Wisconsin, $3.00. Address Alvin G. Koehler, M.D., secretary, Board of Medical Examiners, 46 Washington Blvd., Oshkosh.

Prior to January 1—Arizona, not more than $10.00. Address Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination, 2747 E. McDowell Rd., Phoenix 22.

January 1—California, $20.00 for residents and nonresidents. Address Glen D. Cayler, D.O., secretary-treasurer, Board of Osteopathic Examiners, 1013 Forum Bldg., Sacramento 14.

January 1—Florida, $5.00. Address R. Philip Coker, D.O., secretary-treasurer, Board of Osteopathic Medical Examiners, 602 E. Fourth St., Panama City.

January 1 — Maine, $4.00. Address George F. Noel, D.O., secretary-treasurer, Board of Osteopathic Examination and Registration, Monument Square, Dover-Foxcroft.

January 1—Manitoba, $5.00. Address W. Kurth, D.O., secretary, Board of Osteopathic Physicians, 248 Moorgate Blvd., Deer Lodge, Winnipeg.

January 1—Ontario, $10.00. Address D. G. A. Campbell, D.O., secretary, Board of Directors of Osteopathy, 2 Bloor St. E., Room 22, Toronto 5.

January 1—Saskatchewan, $30.00. Address Doris May Tanner, D.O., secretary, Board of Osteopathic Physicians, 405 Sterling Trust Bldg., Regina.

January 1—Texas, $5.00. Address M. H. Crabb, M.D., secretary-treasurer, Board of Medical Examiners, 1714 Medical Arts Bldg., Ft. Worth.

January 1—Utah, $3.00. Address Mr. Frank E. Lees, assistant director, Department of Registration, 324 State Capitol, Salt Lake City.

January 31—British Columbia, amount of fee set at Annual Meeting of Council of College of Physicians and Surgeons of British Columbia. Address Lynn Gunn, M.D., registrar, Council College of Physicians and Surgeons of British Columbia, 1807 W. 10th Ave., Vancouver 9.

Before February 1—Vermont, $3.00 for residents, $2.00 for nonresidents. Address Charles D. Beale, D.O., secretary, Vermont Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

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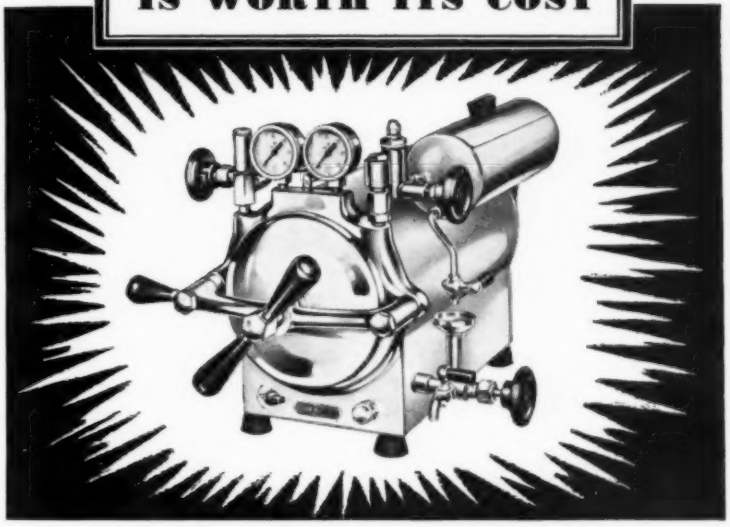

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EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

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Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene; medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given in Philadelphia, Chicago, Kirksville, and Los Angeles under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles, therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

The following Part III examinations are scheduled:

Philadelphia	March 13-14
Kirksville	March 13-14
Chicago	April 3-4
Los Angeles	May 1-2

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1 year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the above named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1 year's internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

TREATMENT OF TUBERCULOSIS*

By J. Burns Amberson, M.D.†

The introduction of streptomycin into the clinical treatment of tuberculosis in 1944 and the subsequent use of other drugs have led to radical changes in the management of this disease. Indeed, there has been no other comparable period in its history. The momentum originally imparted by the discovery of antituberculosis drugs is still felt, and it would be rash to predict when or where it may spend itself, so swift is the rush of events. It seems prudent, therefore, to characterize the following remarks as more of a commentary than a critique.

In the relatively short time since tuberculous patients first received streptomycin, it has been found possible, by adjusting the dosage and combining its administration with that of para-aminosalicylic acid, to circumvent most of its serious toxic properties, as well as to prolong the antimycobacterial effect through many more months than either drug would accomplish alone. There are now a number of other active antituberculosis drugs, the most potent of which are isoniazid and viomycin. Consequently, the variety of drug combinations which may be used simultaneously and in sequence becomes large and the task of assessing their efficacy quite formidable.

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*Reprinted from *Public Health Reports*, October, 1953.

†Dr. Amberson is professor of medicine at Columbia University College of Physicians and Surgeons, attending physician at Presbyterian Hospital, and visiting physician in charge of the chest service at Bellevue Hospital. He received the 1952 Trudeau Medal Award partly for work begun in 1926 and reported in the October 1931 issue of the *American Review of Tuberculosis*. Discussing the failure of a clinical trial of sanocrysin, a gold salt, Dr. Amberson described the criteria he used to judge the clinical effect of this chemotherapeutic agent against pulmonary tuberculosis. His outline of minimum "clinical, laboratory, and X-ray observations sufficient to warrant any reasonable conclusion from such a study" is as valid now as it was in 1931.

An accurate evaluation can be arrived at only after patient and sustained efforts in the laboratory and clinic, which will require years. Eagerness for progress should not be permitted to disrupt the methodical, time-consuming pursuit of scientific facts upon which clinical practice ultimately must stand. In dealing with a problem as complex as tuberculosis, a get-wise-quick approach is thoroughly unsound.

EARLY FAVORABLE EFFECTS

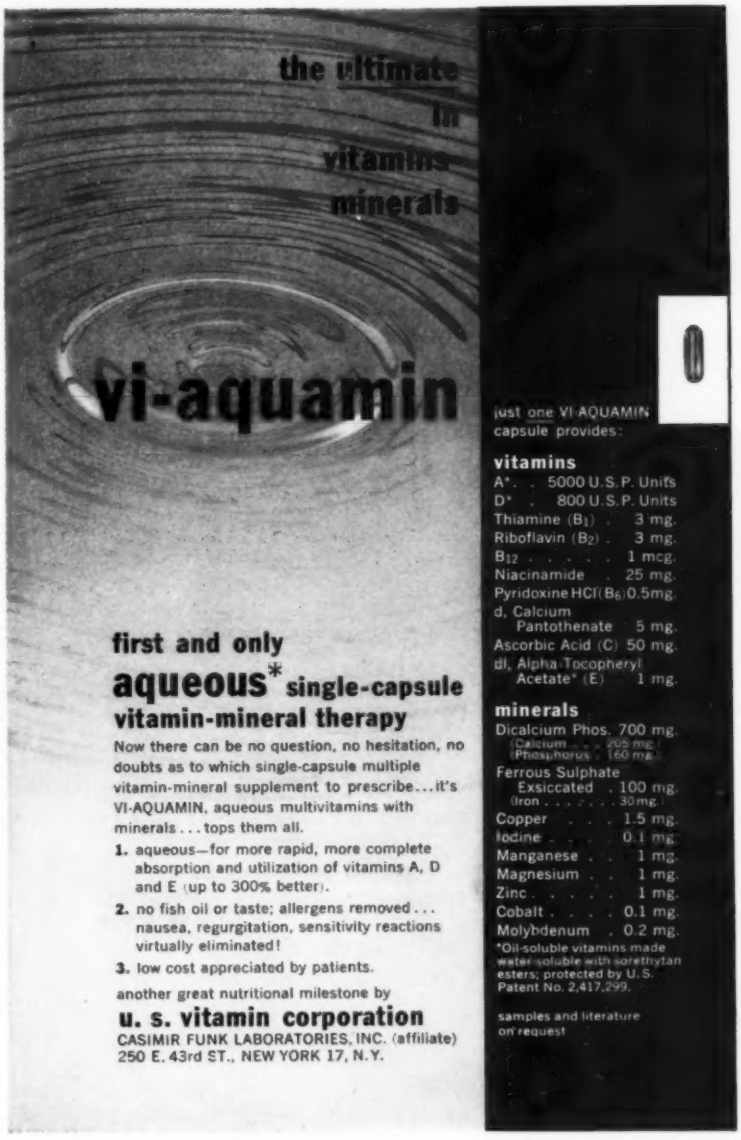
Largely because of the use of specific drugs, the early response of the tuberculous patient to treatment can now be said to be more uniformly favorable, more certainly predictable and, on the average, more rapid than ever before. Whatever the regimen adopted, the first maneuver in treatment is to attempt, as rapidly as possible, to suppress the activity of the tubercle bacillus and thus halt active, progressive disease. The effect of successful treatment is manifested by an abatement of toxemia—allowing the patient to recover his strength and feeling of well-being—and by a gradual restoration of normal physiological processes. Another easily observed effect is the retardation or arrest of active inflammation and destruction of tissue.

The early favorable effects of treatment are often conspicuous. The cough and expectoration may disappear, and drainage from discharging sinuses may dry up completely. The subsidence of inflammation may also be followed by a resolution and absorption of inflammatory products which is remarkable or even dramatic. All this may be observed in the space of 3 or 4 months. But then further regressive changes of the lesions often tend to occur more slowly, and a time may come, perhaps after many more months, when little or no further change is observed, even though the symptomatic recovery is maintained.

Although the early halting of active inflammation and of exudation in and discharge from tuberculous lesions may not be synonymous with the death of the bacteria responsible, these favorable changes nevertheless benefit the patient. At the peak of these effects, necrotic lesions sometimes can be removed surgically without great operative risk. Therefore, one may think of the drying out of lesions as one of the most beneficent, rapid, and striking effects of specific drugs.

DRUG THERAPY VS. REST TREATMENT

The mechanisms of healing under the influence of specific drugs do not appear to be essentially different from those occurring under natural conditions, often favored by a regimen of rest. With minor exceptions, the difference in the early response to treatment does not seem to be qualitative but, rather, a matter of speed and predictability. Thus, the question is raised as to whether these new developments justify or necessitate a change in our ideas regarding the use of rest treatment, which long experience has proved so helpful against tuberculosis.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An answer is difficult because of our limited knowledge of the way in which rest treatment exerts its favorable effects. These effects are vaguely identified as biochemical, hormonal, and general, but not primarily mechanical. Innate factors seem to be vital, and there is reasonable assurance that in most patients these can be raised to better levels by rest treatment and other general measures. If this be so, can specific drug therapy in any way substitute for rest? Certainly, during the early phases of active, progressive tuberculosis, when resistance is low, it seems rational and obligatory to give the patient the benefit of every available help. It is rather generally agreed, therefore, that rest treatment and general adjuvants are just as important as the administration of drugs, and in many cases probably more so.

MEASURING RESISTANCE

Since there is no accurate objective measure of resistance, it is necessary to estimate it almost entirely by clinical judgment based on experience. Although the early response to drug therapy is in most cases consistently favorable, the tendency to relapse after the discontinuance of therapy varies widely in different patients, and this tendency almost certainly is related in part to the level to which resistance has risen. It is much easier, therefore, to identify the time and circumstances initially calling for drugs than it is to identify the time to

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1. Behrman, H. T., Combes, F. C., Bobroff, A., Leviticus, R.: Ind. Med. & Surg. 18:512, 1949.
2. Turell, R.: New York St. J. M. 50:2282, 1950.
3. Heimer, C. B., Grayzel, H. G., and Kramer, B.: Archives Pediat. 68:382, 1951.

stop them. In some patients, an early relapse indicates that chemotherapy has been stopped prematurely; in others, it is continued for an unnecessarily long time, ostensibly for safety's sake.

This dilemma illustrates the practical problem of the physician and his patient, who perforce cannot wait for the laboratory to produce a reliable test of resistance. The best test we have now is the tolerance of the patient for gradually increasing activity, especially physical exercise. And since resistance is the only permanent defense against the infection, it is logical to stop chemotherapy (at a time when healing of the disease is judged to be well advanced) before allowing the patient unrestricted activity, and then gradually to test his tolerance of greater liberty under carefully observed conditions. Justification may be found for speeding his return to a productive life or to more prolonged and radical treatment. This is another one of the problems which require long and careful study.

CONDITION OF TUBERCLE BACILLI

A word should be said about the condition of the tubercle bacilli which remain in tuberculous lesions, particularly those of a solid necrotic character, after the patient has recovered and his disease is called arrested or inactive. It has long been recognized, on the one hand, that the bacteria may survive there for many decades while the patient remains well. Should a relapse occur, these survivors or their descendants are usually found to be the culprits. On the other hand, the bacteria may eventually disappear from the lesions or, if persisting, they may be found incapable of growth on artificial culture media or of causing disease in susceptible animals. A very lively question at the moment is whether the remaining bacilli are really dead, leaving behind only their skeletal remains, or whether they have merely been so enfeebled and modified by the antagonistic forces of nature that they are unable to keep their tenuous hold on life except in the lesions in which they are found.

How long is required under natural conditions for such changes to occur is largely conjectural, but usually it seems to be a matter of many years. It now appears that the continuous administration during many months of combinations of specific drugs such as streptomycin and para-aminosalicylic acid sometimes exerts similar effects on the bacteria. This observation tempts the most critical scientist to entertain the thought that the bacteria may have been killed by the drugs. It is legitimate to pursue the thought and this is being done in various laboratories. Meanwhile, it seems wise and sound to take the position that sterilization of lesions by drugs has not been proved.

TREATMENT PRINCIPLES

In sum, it may be said that even though the patient profits greatly from the use of specific drugs when they are needed and may experience an early halting of serious disease which in earlier days almost always led to chronic illness at best and early death at worst, his ultimate prospects of permanent recovery depend largely on other help which may be given him. Particularly they depend on the effect of rest treatment to build up his resistance for the long battle against the tubercle bacilli which survive in his tissues.

If resistance against infection has risen to a high level during treatment and the residuals of disease are small, the prognosis for permanent recovery is excellent. The recovery under drug treatment of patients whose resistance is poor usually is followed sooner or later by relapse. However, surgical removal of the grossly damaged tissue, if practicable, may effectively turn the balance in such a patient's favor. Obviously, therefore, each case must be considered according to its individual features.

Certainly, a most important principle is to treat the patient with recently developed and limited tuberculosis promptly and sufficiently long to prevent extensive destruction of tissues and chronic disease and to insure the best prospect of permanent recovery. Many of these patients have no symptoms, and their disease is discovered in case-finding surveys or in other routine examinations.

Although the rest cure alone has been effective in a great majority, the time required has been long, often a year or more. Now, although recovery may not be speeded greatly by drugs, their use when needed should help assure a permanent arrest of the disease almost uniformly; only a few of such patients will require surgery.

In the more advanced and active cases, reliance is now placed chiefly on the immediate and prolonged use of combined drug therapy to gain early control of the infection and to permit resolution of inflammatory changes. Mechanical procedures such as thoracoplasty and artificial pneumothorax are usually reserved for mechanical problems such as residual unhealed cavities in the lungs. Similarly, surgical resection is preferably reserved for the time when the disease has been brought under control and the remaining problem is the threat of relapse. During this time of waiting, it is to be hoped that the resistance of the patient may rise to a maximum level under treatment so that the permanence of his recovery may be improved or assured.

Another debatable question is the place of the so-called ambulatory treatment. If this could be shown to be adequate, the obvious advantages are that the huge cost of hospital care would be reduced, the patient's morale possibly would be improved, and the patient would not suffer the usual heavy social and economic losses. It is questionable whether these theoretical advantages can be achieved without paying the price of frequent relapse or of long chronic disease leading to disability and eventually to death. Certainly, the matter should be approached objectively. Almost every conceivable treatment of tuberculosis has been applied in the past to the ambulatory patient. The early favorable response frequently is deceptive, and the opportunity to bring about a permanent recovery is easily lost.

PATIENTS AS SOURCES OF INFECTION

Tuberculous patients with long-established chronic disease who circulate and act as sources of infection in the community represent a serious situation everywhere. As life is saved or prolonged by treatment, the death rate drops but the number of living patients continues to be high, and in fact, rises in some groups, particularly elderly men. Since many of these patients are destined for continuing chronic and eventually fatal disease, the problem of eliminating the source of infection short of the death of the patient must be considered. Giving drugs and thus drying up pulmonary cavities appeals to the imagination as a practical public health measure, and studies of this possibility are under way.

Here again, the only reasonable attitude is an objective one. Thus far, no combination of drugs has proved permanently adequate in preventing open cavitary lesions from discharging bacteria. Furthermore, when a dry cavity again becomes wet and starts discharging and the patient again starts coughing, he may be spreading drug-resistant strains which, when they infect a previously uninfected person, may produce disease which will not be amenable to drug therapy. The hope would be to circumvent this by resorting to different series of drugs, but the practical feasibility of this course is at best open to debate.

PROSPECTS FOR THE FUTURE

In view of our better understanding of the various modes of treatment and of the outlook for the discovery of new drugs, the future holds brightening prospects for the tuberculous patient. To indulge in the hope of eventually finding an absolute and final cure of the disease would be idle at this time. The methods of study and investigation promising to lead to further knowledge are well known, but the time they require is necessarily long. One thing is certain, though: Progress will be made in laboratories and clinics like those of the Henry Phipps Institute, and the scientists who will achieve that progress will be of the same sterling character as that group at the Phipps which has given it such an illustrious past. We place in their hands, and in those of similarly competent and devoted workers, the privilege and responsibility of coming to grips with the almost limitless problems that remain.

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THE CURRENT DIABETES PROBLEM*

Diabetes remains a disease of major medical and public health interest. Of the chronic diseases common in middle and later life, it is not only one of the easiest to diagnose but it also offers the best prospects of control in the individual patient. As a result of progress in the medical management of the disease and the introduction of insulin therapy, the longevity of diabetics has steadily increased, but this in itself has brought some new problems.

Among white persons the death rates are consistently below 1 per 100,000 at the ages under 15 and are less than 2 per 100,000 up to age 35. Past midlife, however, the rate increases rapidly with advance in age, much more so among women than among men. As a consequence, the death rate from diabetes at ages 55 and over is about twice as high for women as for men.

Colored persons now suffer a higher death rate from the disease than do the whites. Among males the margin is slight, but for females the colored rate is the higher by about one fifth. However, the excess mortality among the colored is found only up to age 55 for men and up to age 65 for women. These facts relate to an essentially urban population which is within the reach of the best medical facilities and in which the living and working conditions of the colored are approaching the average community level. In the general population of the United States, the death rate from diabetes among white persons still exceeds that among the colored.

The list of causes of death among diabetics is becoming more and more like that among nondiabetics. This is evident from an analysis, completed recently by the Metropolitan Life Insurance Company, of the experience of the many thousands of diabetic patients treated at the Joslin Clinic in Boston. During the period from January 1, 1950, to May 5, 1953, three fourths of the 1,358 deaths recorded were due to cardiovascular-renal diseases. A major portion of deaths in this broad category was accounted for by heart disease of the coronary type, a condition which occurs earlier and develops faster in diabetics than in nondiabetics.

The second largest cause in this category, accounting for nearly one eighth of the deaths from all causes, was renal disease. Most of these fatalities were due to a serious complication—diabetic nephropathy—which is now perhaps the most important numerically among the causes of death peculiar to diabetes. A high proportion of deaths from this cause occurs among patients with long duration of the disease which began in childhood or young adult life. To that extent it is a relatively new medical problem in diabetes and, because of its severity, is a source of major concern. Of approximately the same frequency as renal conditions were deaths of cerebral origin. In contrast, gangrene, formerly a frequent and dreaded complication of diabetes, caused only 1.3 percent of all deaths in recent years.

Death from infection has been reduced to minor importance in the total mortality among diabetics, reflecting the generally improved condition of persons with the disease and the benefits attending the use of the antibiotics and chemotherapy. Pneumonia and other nontuberculous respiratory diseases accounted for 4.0 percent of the deaths, and tuberculosis for only 1.0 percent. Notable also are the low proportions of deaths from diabetic coma and insulin reactions. Diabetic coma, which, before insulin, was the major cause of death among diabetics, was responsible for only 1.0 percent of the recent deaths. Even among patients with onset in childhood, only 6.7 percent of the deaths were due to this cause. Fatal insulin reactions were few, accounting for only 3 out of the 1,358 deaths.

In the recent period, cancer was responsible for 10 percent of the deaths among patients of the Joslin Clinic. The actual death rate from cancer among diabetics is not very different from that in the general population.

The predominance of the cardiovascular-renal diseases in the mortality picture among diabetics reflects primarily their longer survival and hence their

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, September, 1953.

increasing average age at death. Life tables based upon the experience of the Joslin Clinic show that at every age, and more especially at the younger ages, the expectation of life of diabetics has increased substantially. No less than 35 percent of the 1,358 recently deceased patients had lived 20 years or more after the onset of their disease, a far better survival experience than was recorded less than two decades ago.

Further gains in longevity of diabetics are in prospect. These patients will benefit from the fruits of research into the disease and into insulin, as well as from advances in the field of medicine generally. Beyond that, gains of major proportions are possible by greater efforts to diagnose the disease early and to improve the general level of treatment of diabetics. The toll of premature death from diabetes because of neglect of treatment is much greater than it need be. Patients and physicians working as a team can do much to reduce the preventable mortality from the disease.

MILLIONS OF MOTHERS EMPLOYED*

Just over 5¼ million women workers were mothers of children under 18 in April, 1951, according to the Bureau of the Census; about 2 million had children under 6.

Absence of the husband tends to force mothers into seeking employment. Of all mothers with children under 18, one-fifth of those with the husband present and one-half of all other women that were ever married were employed.

Even among mothers whose children were all under 6, the proportion of working mothers was considerable—14 percent for those with husband present, and 37 percent for all others ever married.

*Reprinted from *The Child*, April, 1953.

A REVIEW OF POLLENOSIS AND THE ROLE OF WEEDS*

By W. C. Spain, M.D.†

(Continued from the November JOURNAL*)*

THREE SEASONAL GROUPS

In the Northeastern area of the United States the dates of pollination of the important hay fever and asthma producing plants permit a sharp division of the pollen victims into three groups. In the first are those whose symptoms occur between mid-March and June first. No weed is a culprit although many tree pollen victims in this group are ready to stigmatize as weeds the real sources of their discomfort, which are the ash, beech, birch, elm, oak, hickory, paper mulberry, and poplar. The pollen of the alder and of the swamp sedges occasionally produces symptoms.

In the second group, the symptoms persist from mid-May to mid-July. English plantain (*Plantago lanceolata*) is a weed of much importance here, with sorrel (*Rumex acetosella*) of lesser importance. Of greater moment than these weeds, however, is the family of grasses —timothy, orchard, oat, rye, redtop, june, bermuda, sweet vernal, velvet. Roses, since insect pollinated, are innocuous unless cut and kept indoors. The term "rose cold" is, therefore, an incorrect designation for the summer type of hay fever.

In the third group of patients the symptoms occur from mid-August to frost, and it is here that the weed asserts its importance. The ragweeds, giant and dwarf (*Ambrosia trifida* and *elatior*), are the chiefs of them all, having the dubious reputation of being the cause of more suffering than all other pollens combined. Not only does the discomfort they produce involve a greater number of victims, but the suffering is more intense in degree and occurs at a time of the year, at the threshold of autumn, when secondary, complicating bronchial and sinus diseases are encouraged to appear. It has been estimated that one-third of all untreated ragweed hay fever sufferers eventually develop bronchial asthma, a much more serious and disabling disease. Of lesser importance than ragweed are cocklebur

*Reprinted from *Public Health Reports*, September, 1953.

†*Dr. Spain is professor of clinical medicine at the Post Graduate Medical School, New York University, and attending physician and director of the division of allergy, medical department, University Hospital. He is a member of the editorial board of the Journal of Allergy. This paper is condensed from an original lecture given by the author at the weed control conference conducted by the New Jersey State Department of Health at Rutgers University on April 8, 1953.*

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(*Xanthium*); lambsquarters (*Chenopodium*); pigweed (*Amaranthus*); mugwort (*Artemisia*); American hemp; wild rice (*Zizania*); great reed (*Phragmites*); marsh-elder (*Iva*). Usually goldenrod has on it some adherent ragweed pollen, deposited by wind from adjacent ragweed but, as stated, goldenrod does not deserve the evil reputation it has. Its pollen, since insect borne, will cause no symptoms unless the blossoms are brought indoors.

The list of weeds which produce allergic discomfort is even greater in other areas of the United States. In the plains States, southwestern States, and the Pacific States, the most troublesome are the chenopods, thistle(*Salsola*), and burning bush (*Kochia*), wormwood and sagebrush (*Artemisia*).

AREAS FREE OF POLLEN

Since the distressing allergic symptoms result from actual physical contact of pollen and patient, the surest way for the patient to obtain relief is to escape to an area where the pollen producing plants particularly disturbing to him do not grow. Bermuda, Nova Scotia, the tropics, our own southwest and areas in the Rocky Mountains offer to ragweed sufferers complete freedom from hay fever. Less completely free localities are the southern tip of Florida, California, areas in the heavily wooded sections of the upper Michigan peninsula, of Maine, and of Canada and some parts of the White and of the Adirondack Mountains. The pollen surveys of the New York State Department of Health have established the relative freedom from pollen of numerous Adirondack localities (*1*).

METHODS OF DESTROYING THE WEEDS

Such escapes from pollen, however, are impractical or impossible for the majority of sufferers, who cannot be absent from their work or their families for the long intervals required. For them relief can be expected by attacking the hay fever producing weeds themselves by eradication, a slow, painful, and not very successful process, by manual removal or by cutting at the strategic moment when pollination is imminent.

Far superior are the chemical methods. New York City, according to information from its department of health, and several New Jersey communities conducted in 1946 (*2*) the first centrally directed ragweed control spraying program. By employing a spray of the hormone 2, 4-D, dichlorophenoxyacetic acid, it has been possible greatly to reduce the growth of ragweed within the city limits. In proper dilutions it is reported to be selective in action in that it does not kill desirable grasses, but it is known, of course, to be lethal for vegetables and flowers (*5, 6*). Persistence in spraying the ragweed areas each year seems essential to prevent a return of the ragweed. Following the

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example of New York City, other municipalities have adopted this plan of extermination. Until all States in the ragweed zone collaborate in a determined and extensive plan, however, the hay fever victim may be somewhat benefited, but will continue to suffer, since the pollen produced by weeds many miles away can be easily transported to him by air currents.

INDIVIDUAL'S CONTROL OF POLLEN

The patient can conduct a plan of weed pollen control in his immediate environment by the installation in his bedroom or home, and in his place of business, of efficient conditioning units. Such units should filter but not chill the air, since respiratory membranes irritated and congested by pollens seem especially prone to "colds," acute respiratory infections, or sinusitis when suddenly subjected to excessively chilled air. Too, to be most effective, conditioned areas should have as their source of outside air only the conditioning apparatus, and all doors or windows should be kept closed; as little traffic as possible should be permitted into the area, since disturbing quantities of pollen may be imported upon the hair and clothing of those entering.

These measures of escape and avoidance are not altogether successful. Efforts must be made to so condition the pollen sufferer that he may continue to work and live in the pollen-containing atmosphere. This is not the time or place to discuss the therapy of hay fever —but it is pertinent to say that such treatment is based upon an attempt to increase the tolerance of the sensitized cells so that they do not react with such vigor or intensity upon exposure to the disturbing pollen. Minute, gradually increasing amounts of an aqueous extract of the specific pollen excitant are given hypodermically at weekly intervals during the period beginning 3 months before the expected season and extending through it; or, in selected cases, larger doses administered once monthly throughout the year, though once weekly during the season, are effective (*7*). The use of antihistaminic drugs, of ephedrine, of soothing eye drops, of avoidance of dusts, gases, and chemical fumes contribute to the relief of the patient.

The most important step in the handling of any allergy problem is the attempt to remove the cause. This step can often be readily accomplished by the allergist if the exciting substance, identified by various tests, be an animal dander such as that of the cat or horse, causing asthma; or a food such as egg or chocolate, causing asthma or urticaria. It is impossible to accomplish if the exciting substance cannot be readily removed, being derived from widespread

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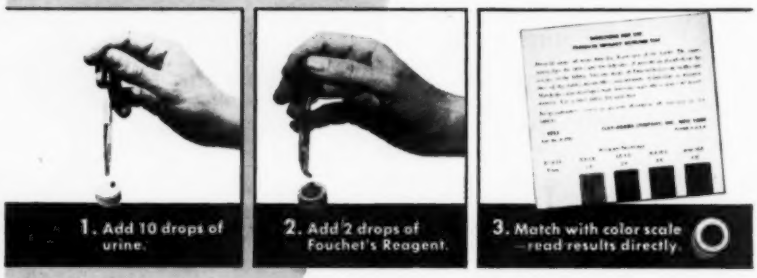

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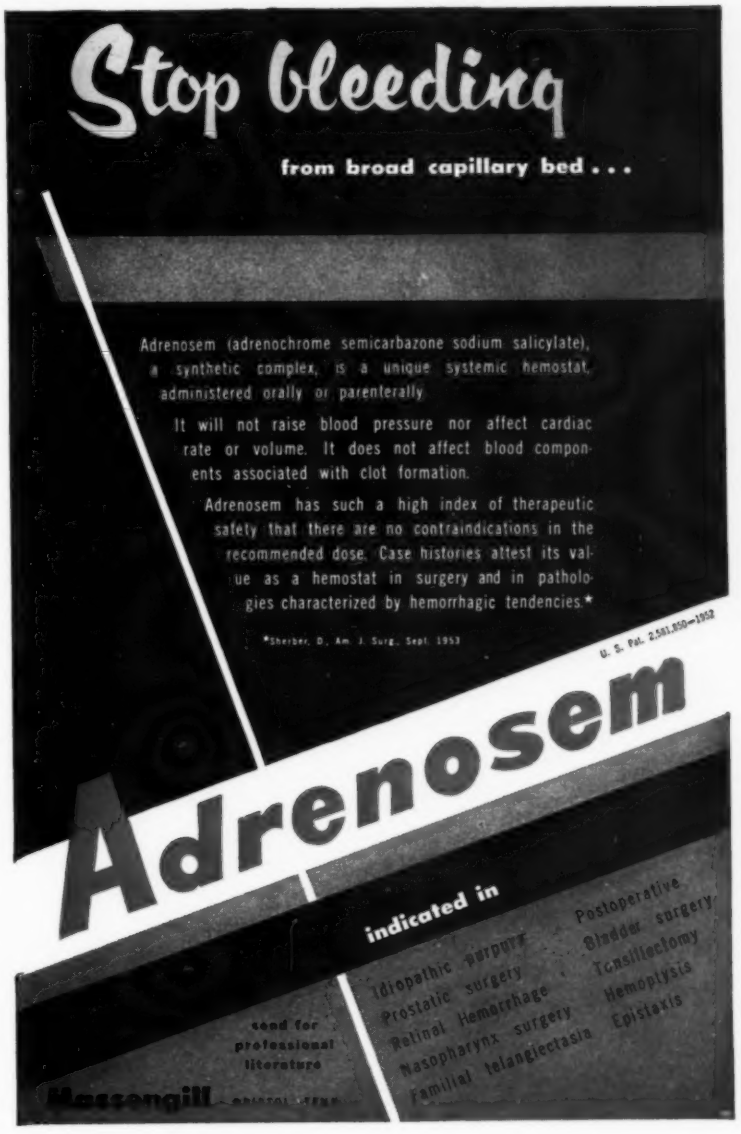
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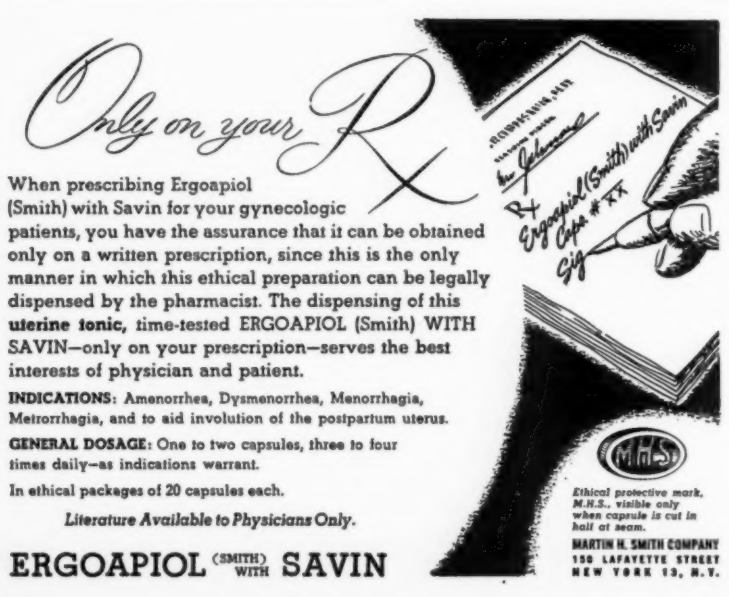
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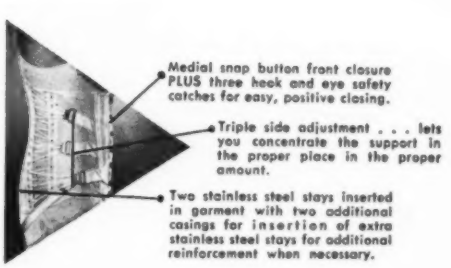
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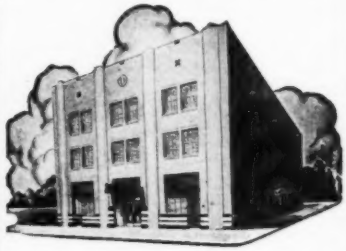
GASTRO-INTESTINAL

Special attention to resistant colon and rectal conditions



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increased uric acid excretion

in both acute and chronic stages

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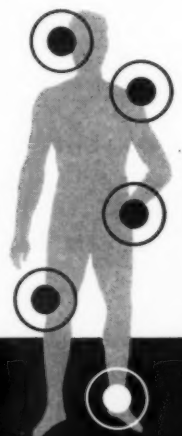

Each *Entab** (enteric-coated tablet) contains:

Sodium Salicylate 0.25 Gm. (4 gr.)
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Colchicine 0.25 mg. (1/250 gr.)

SUPPLIED:
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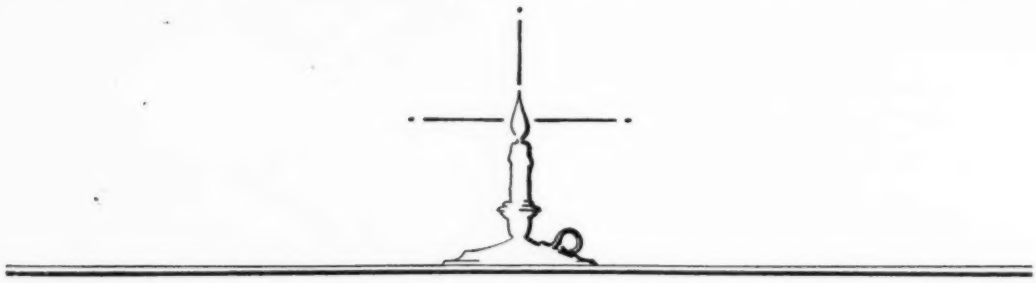
A BETTER LIGHT FOR DIOGENES



IOGENES was a Greek, and a philosopher. He belonged to the school of cynics. Which means that he associated with a group of men who didn't believe in practically anything.

Now among the many things Diogenes didn't believe in was an honest man. But he wanted to give every one a chance, so he roamed the highways and byways of Athens with a lantern which he would flash on the faces of the passers-by, endeavoring to find in their features some sign of integrity.

Nothing ever came of his labors. (According to him). But because he was a philosopher and a reasonably fair man, Diogenes admitted that it *could* be his lantern. And so, he complained, "If only I could have a better light . . ."

And many people tried to give Diogenes a better light . . . If you're as old as sixty, you may remember that a fellow named Welsbach thought up a contraption that you fitted over a gas-jet . . . Then there was that young fellow, Tom Edison, who worked out the electric light bulb. And another genius planned a new type of light called fluorescent . . . But none of them succeeded in inventing a light that helped Diogenes.

Then a man comes up with still another kind of light—a poor sort of thing. It was a little blob of wax, wrapped around a piece of string. And one Christmas Eve he took it over to Diogenes' home. There the old fellow was, visiting with his cynic cronies, and saying as how "No, definitely not! There just wasn't such a thing as an honest man." And the cynics all nodded their heads in agreement. Then the man came in with his candle, and he lighted it. And Diogenes could hardly believe his eyes! Because as the soft, mellow beams shone out from the Christmas candle and fell on the faces around him, Diogenes could see, just as plain as could be, that there wasn't a really dishonest man in the entire gathering.

And ever since then, at Christmas time, the little Christmas candles have made fools out of cynical people by proving that it is seldom that you can find a human being who, 'way down deep inside, isn't essentially honest . . . if you'll only look at him in the proper light.

From a fantasy by K. Miles

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can find a lot of comfort in a tube of
V-E-M
Nose Ointment
with
APPLICATOR

Comfort from the irritation of
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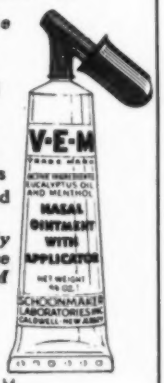
- Bad breath exhaled through the nose

Children especially react favorably to use of the handy V-E-M Applicator.



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. . . for muscle soreness and stiffness lumbago and neuritis is discomfort

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- no deleterious side actions—no drug-induced allergic reactions...
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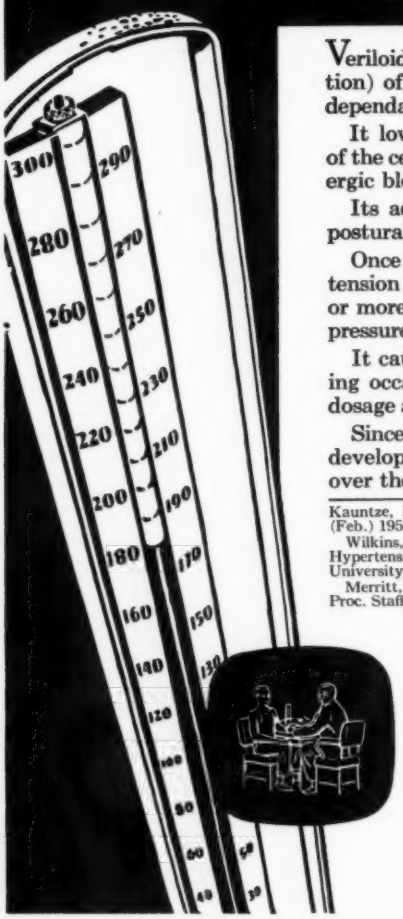

Veriloid, the selective alkaloidal extract (alkavervir fraction) of Veratrum viride, biologically assayed in dogs, is dependably uniform in potency and action.

It lowers blood pressure promptly through mediation of the central nervous system, without ganglionic or adrenergic blockade. Not a local vasodilator.

Its administration is not fraught with the dangers of postural hypotension.

Once dosage is established, prolonged control of hypertension is possible. Even after nine months' therapy 30% or more of patients retain the induced reduction in blood pressure—a notably high percentage.

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Since action is not cumulative, since tolerance does not develop, Veriloid is excellently suited "for the long pull" over the years.

Kauntze, R.: Veratrum and Its Derivatives, The Practitioner *170*:189 (Feb.) 1953.

Wilkins, R. W.: Recent Experiences with Pharmacologic Treatment of Hypertension, in Bell, E.T.: Hypertension, A Symposium, Minneapolis, University of Minnesota Press, 1951, p. 492.

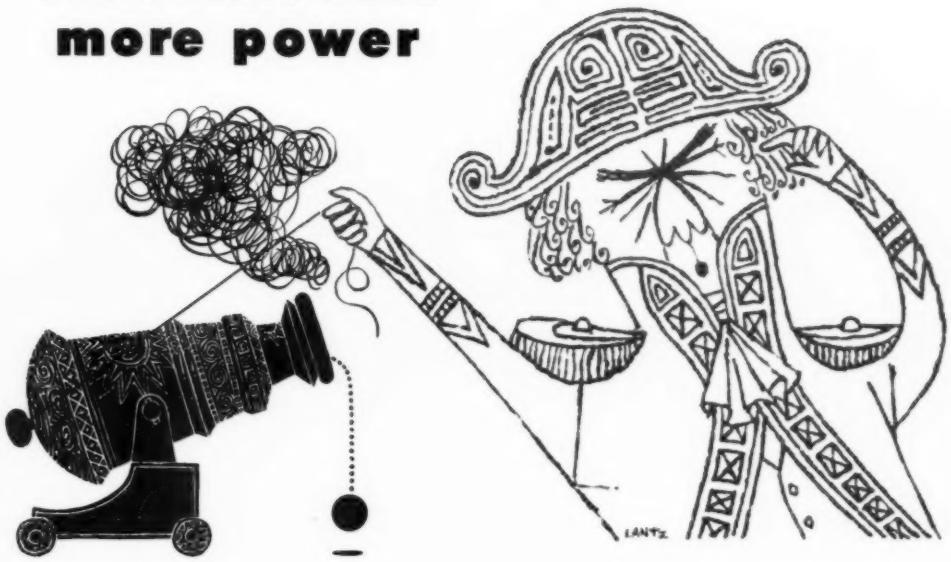
Merritt, W. A.: The Treatment of Essential Hypertension with Veratrum, Proc. Staff Meet. Mayo Clin. *27*:481 (Nov. 19) 1952.

RIKER LABORATORIES, INC.
8480 Beverly Boulevard • Los Angeles 48, California

VERILOID is supplied in slow-dissolving scored tablets, in 2 and 3 mg. potencies. Initial recommended dosage, 9 mg. daily, in divided doses, not less than 4 hours apart, preferably after meals. Maintenance dose, 9 to 24 mg. per day.

VERILOID-VP provides in each scored tablet 2 mg. of Veriloid and 15 mg. of phenobarbital. Initial daily dosage, 1 or 2 tablets t.i.d. or q.i.d.

some cases need
more power



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1. *Spies, T. D.: Influence of Pregnancy, Lactation, Growth, and Aging on Nutritional Processes, J.A.M.A., 153:185 (Sept. 19), 1953, p. 189.*

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VITAMIN B_{12}	5 mcg.	IRON	10.0 mg.
VITAMIN B_1	10 mg.	MAGNESIUM	6.0 mg.
VITAMIN B_2	5 mg.	MANGANESE	1.0 mg.
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CALCIUM	103.0 mg.	POTASSIUM	5.0 mg.
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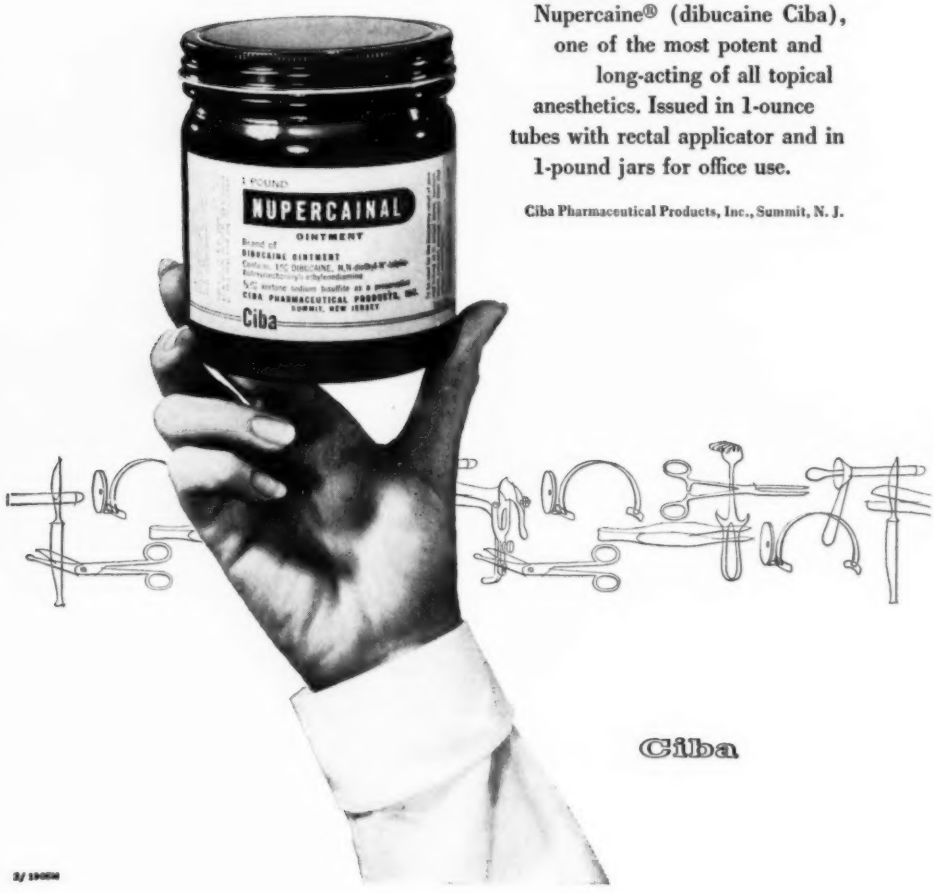

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